Dermatology

>> UW tables & charts in the same sequence of FA

>> + Some added notes

Dedicated to my wonderful sister..... amooon

Dermatology

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Dermatology

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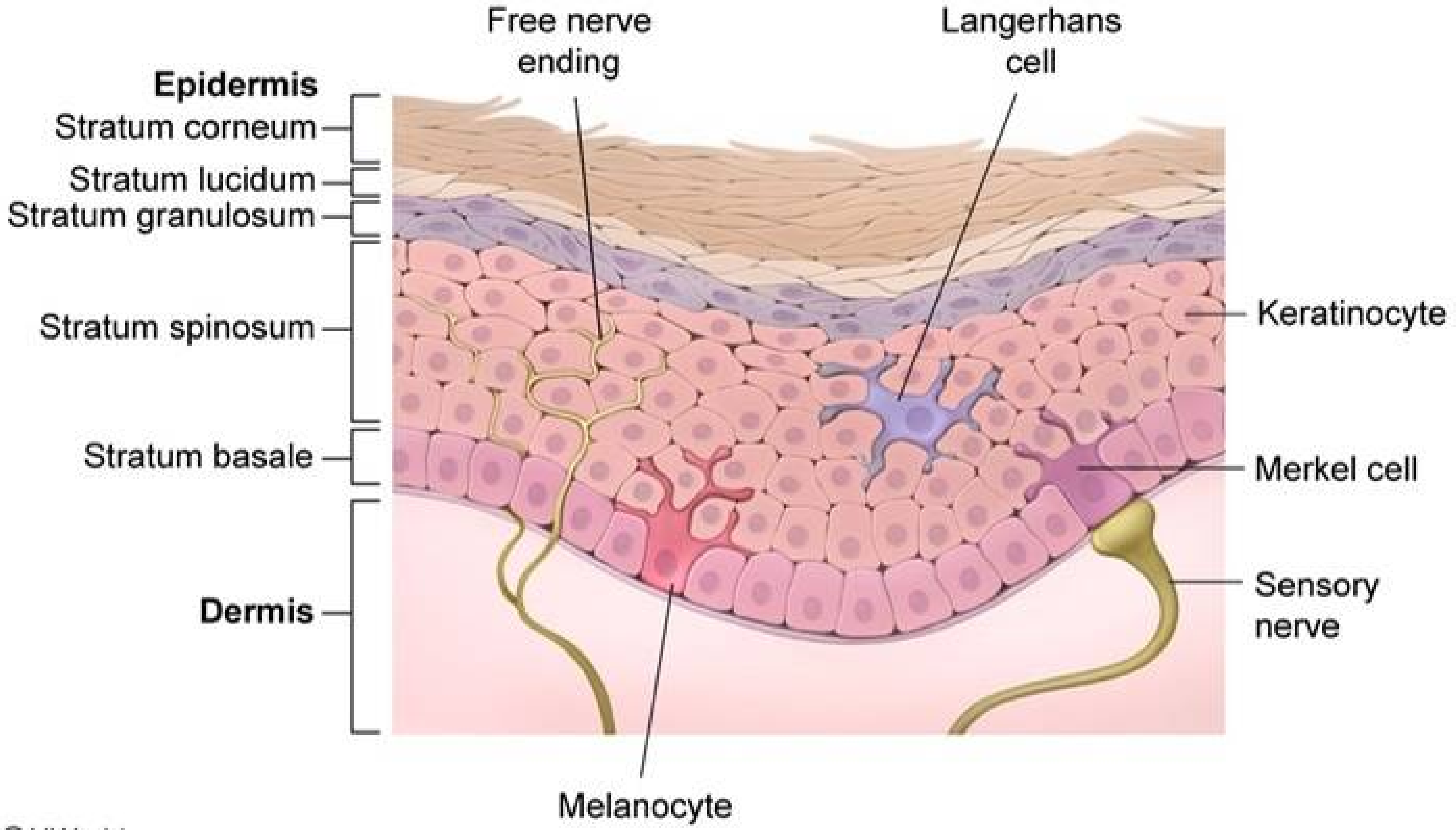
Layers of the epidermis

Come, Let's Get Sun Burned



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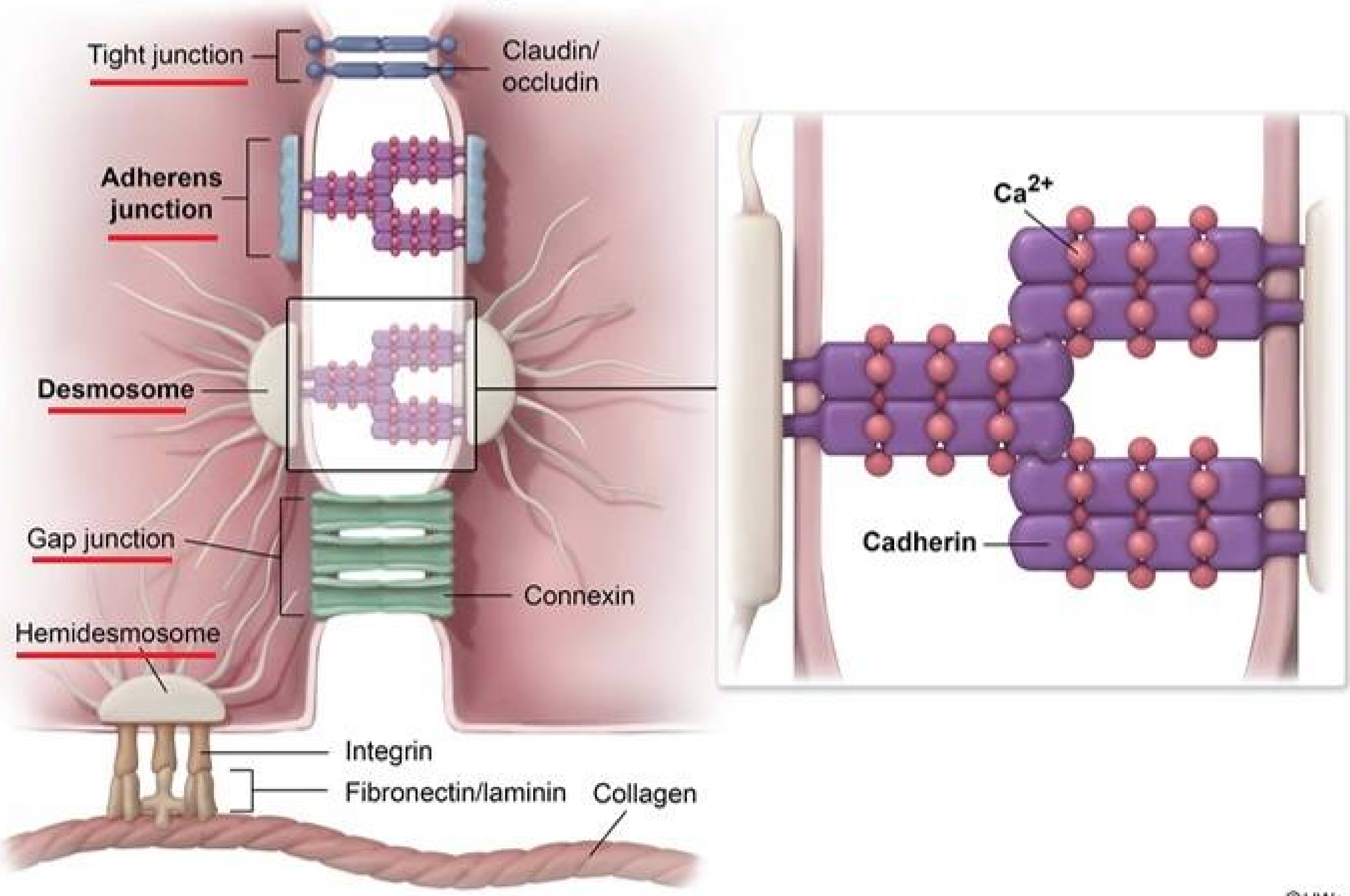
Cell junctions and cadherins



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Cell junctions		
Type	**Proteins**	**Function**
Gap junction	Connexins	Intercellular communication
Tight junction	Claudins, occludin	Paracellular barrier
Adherens junction	Cadherins	Cellular anchor
Desmosomes	Cadherins (eg, desmogleins, desmoplakin)	
Hemidesmosomes	Integrins	

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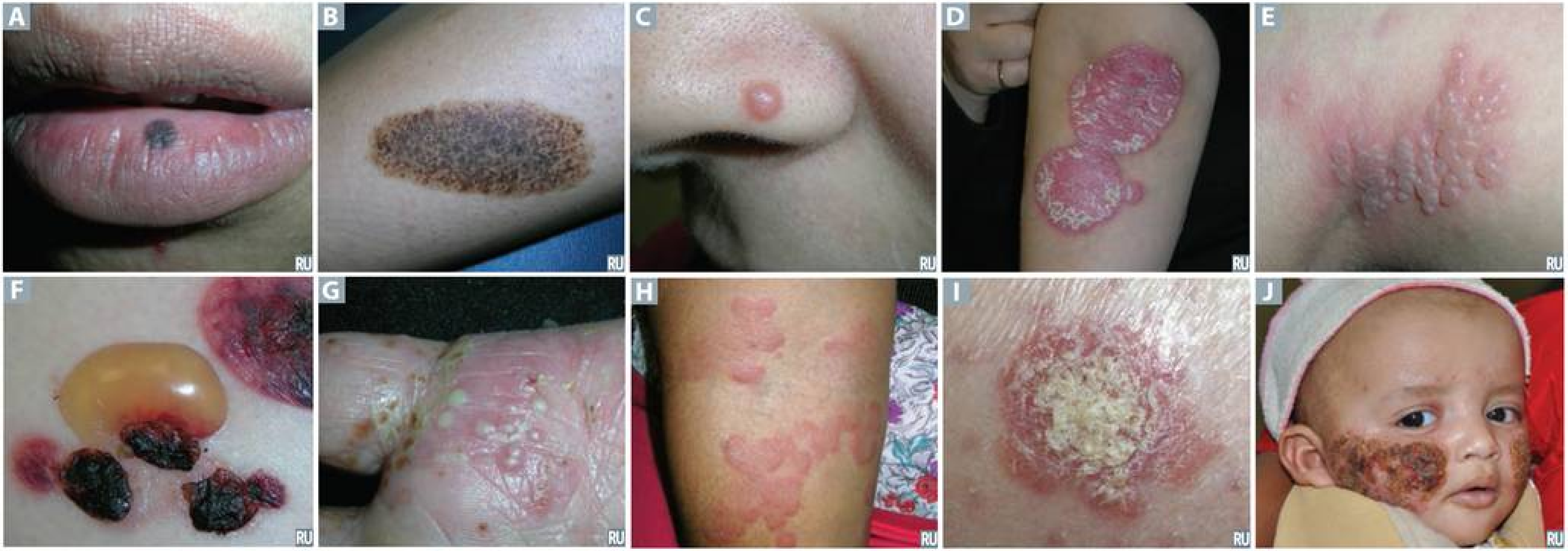
Dermatologic macroscopic terms

LESION	CHARACTERISTICS	EXAMPLES
Macule	Flat lesion with well-circumscribed change in skin color < 1 cm	Freckle (ephelide), labial macule A
Patch	Macule > 1 cm	Large birthmark (congenital nevus) B
Papule	Elevated solid skin lesion < 1 cm	Mole (nevus) C, acne
Plaque	Papule > 1 cm	Psoriasis D
Vesicle	Small fluid-containing blister < 1 cm	Chickenpox (varicella), shingles (zoster) E
Bulla	Large fluid-containing blister > 1 cm	Bullous pemphigoid F
Pustule	Vesicle containing pus	Pustular psoriasis G
Wheal	Transient smooth papule or plaque	Hives (urticaria) H
Scale	Flaking off of stratum corneum	Eczema, psoriasis, SCC I
Crust	Dry exudate	Impetigo J


A
B
C
D
E
F
G
H
I
J
RU

Purpura>> **4 to 10 mm in size**

Petechiae>> <4 mm in size

Ecchymosis>> >1 cm in size

Dermatologic microscopic terms

LESION	CHARACTERISTICS	EXAMPLES
Hyperkeratosis	↑ thickness of stratum corneum	Psoriasis, calluses
Parakeratosis	Retention of nuclei in stratum corneum	Psoriasis, actinic keratosis
Hypergranulosis	↑ thickness of stratum granulosum	Lichen planus
Spongiosis	Epidermal accumulation of edematous fluid in intercellular spaces	Eczematous dermatitis
Acantholysis	Separation of epidermal cells	Pemphigus vulgaris
Acanthosis	Epidermal hyperplasia (↑ spinosum)	Acanthosis nigricans, psoriasis

Albinism>>

Normal melanocyte number with decreased melanin production due to decreased tyrosinase activity or defective tyrosine transport.

Increased risk of skin cancer.

Melasma (chloasma)>>

Acquired hyperpigmentation associated with pregnancy (mask of pregnancy) or OCP use.
More common in women with darker complexions

Vitiligo	
Clinical manifestations	• Depigmented macules on acral areas & extensor surfaces; face commonly affected • Lesions may be symmetrical, dermatomal, or unilateral
Clinical course	• Most cases progress gradually • Repigmentation is spontaneous in 10%-20% of cases • Increased incidence of other autoimmune disorders (eg, lupus, thyroid disease, pernicious anemia, Addison disease)
Treatment	• **Limited disease:** Topical corticosteroids • **Extensive/unresponsive disease:** Oral corticosteroids, topical calcineurin inhibitors, PUVA

PUVA = psoralen + ultraviolet A light.

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Vitiligo

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Seborrheic dermatitis	
Clinical features	• Peaks in infancy & adulthood • associated with colonization by *Malassezia* • **associated with Parkinson disease** • Erythematous plaques &/or yellow, greasy scales • Located on scalp, face (eg, eyebrows/eyelids, posterior ears, nasolabial folds), umbilicus, diaper area
Treatment	• First-line: Emollients, nonmedicated shampoos • Second-line: Topical antifungals or low-potency glucocorticoids

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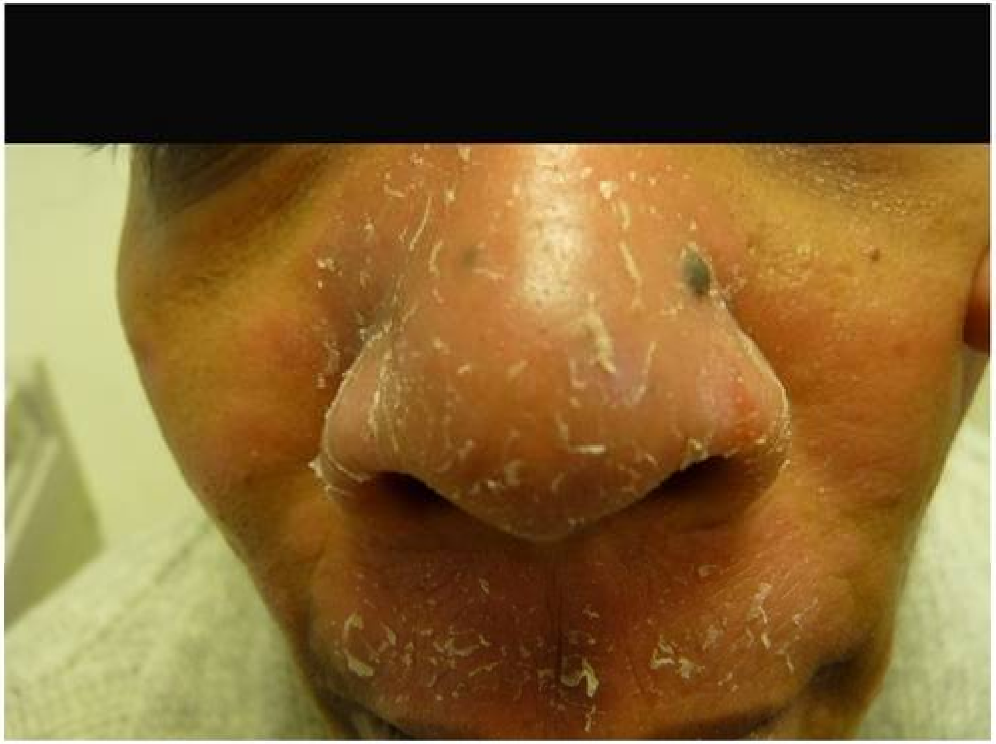
Cradle cap

Seborrheic dermatitis>>



Acne vulgaris	
Clinical features	• **Comedonal acne:** closed or open comedones on forehead, nose & chin • **Inflammatory acne:** small, erythematous papules & pustules • **Nodular acne:** large, painful nodules; sinus tracts & scarring
Pathogenesis	• Hyperkeratinization & obstruction of pilosebaceous follicle • Sebaceous gland enlargement & increased sebum production • Metabolism of sebaceous lipids by *Cutibacterium acnes* & release of inflammatory fatty acids • Follicular inflammation & rupture
Risk factors	• Increased circulating androgens (eg, puberty, polycystic ovary syndrome) • Mechanical trauma/friction (eg, excessive scrubbing, tight clothing) • Comedogenic oil-based skin & hair products • Excessive heat • Obesity

Acne vulgaris>>

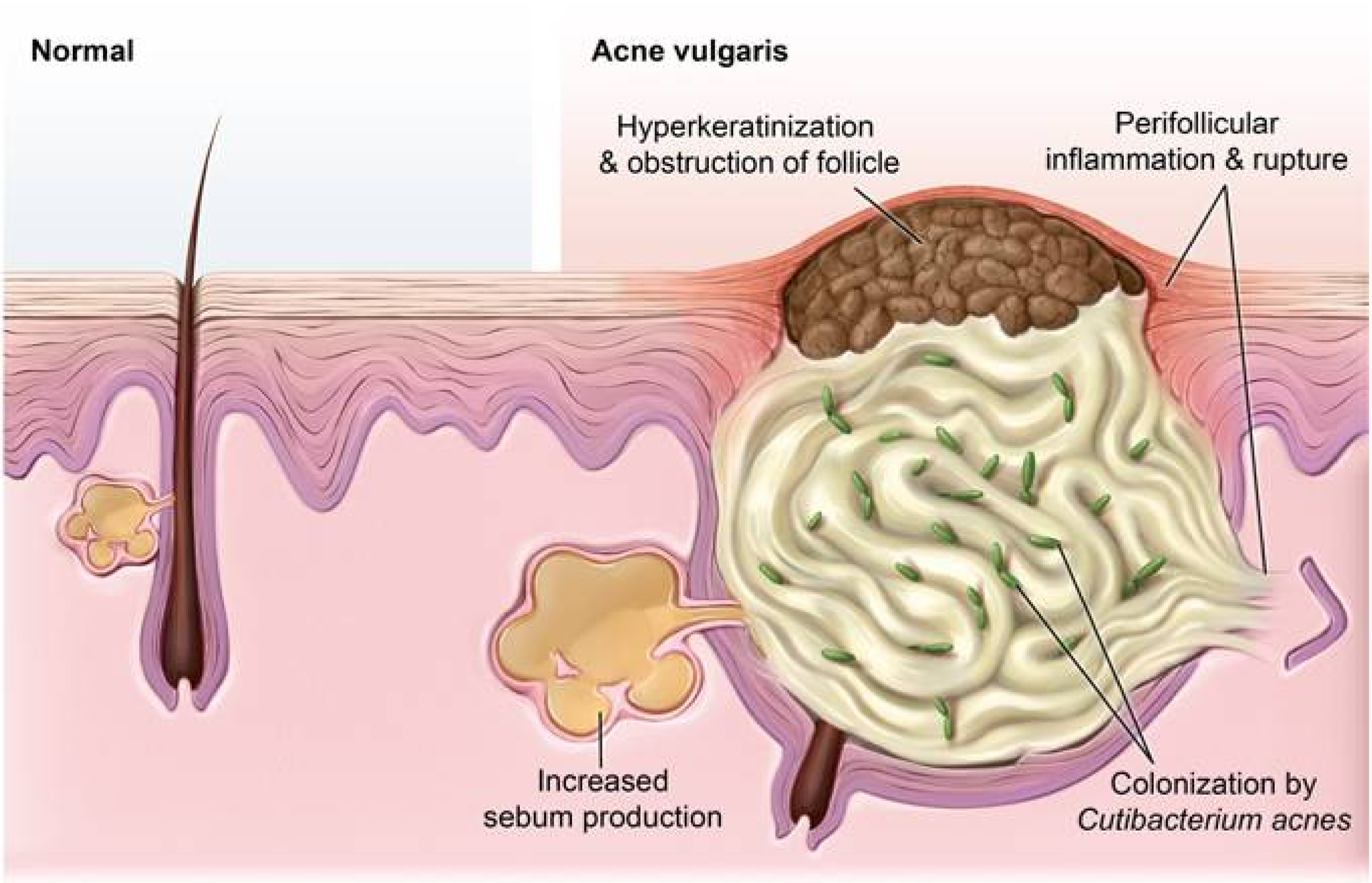
closed comedones (whiteheads)

open comedones (blackheads)

Inflammatory acne>>

Nodulocystic acne>>

Pathogenesis of acne



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Treatment of acne vulgaris	
	Comedonal acne • Closed or open comedones on forehead, nose & chin • May progress to inflammatory pustules or nodules • Treatment: **Topical retinoids**; salicylic, azelaic, or glycolic acid

	Inflammatory acne
	• Inflamed papules (<5 mm) & pustules; erythema
	• Treatment:
	  • Mild: Topical retinoids + benzoyl peroxide
	  • Moderate: Add topical **antibiotics** (eg, erythromycin, clindamycin)
	  • Severe: Add oral antibiotics

	Nodular (cystic) acne
	• Large (>5 mm) nodules that can appear cystic
	• Nodules may merge to form sinus tracts with possible scarring
	• Treatment:
	  • Moderate: Topical retinoid + benzoyl peroxide + topical antibiotics
	  • Severe: Add oral antibiotics
	  • Unresponsive severe: **Oral isotretinoin**

Side effects of oral isotretinoin therapy
• Teratogenic (eg, spontaneous abortion, fetal malformations) • Hyperlipidemia • Chelitis, dry skin • Myalgias • Pseudotumor cerebri

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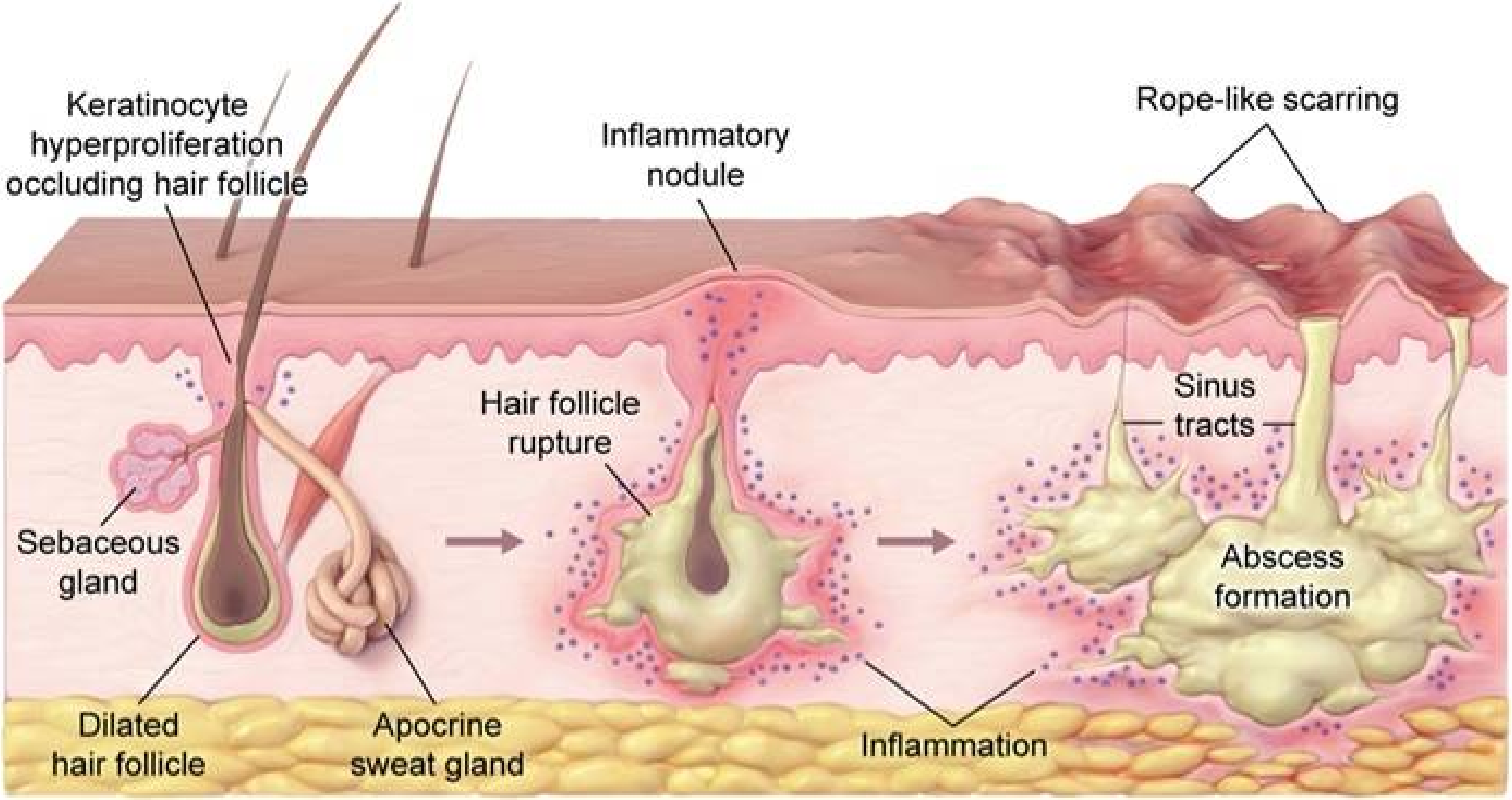
Drug-induced acne	
Common triggers	• **Glucocorticoids**, androgens • Immunomodulators (eg, azathioprine, EGFR inhibitors) • Anticonvulsants (eg, phenytoin), antipsychotics • Antituberculous drugs (eg, isoniazid)
Presentation	• **Monomorphic papules** or pustules • Lack of comedones, cysts & nodules • Location & age of onset may be atypical for acne
Management	• Discontinue offending medication • Standard acne therapy unlikely to be effective

EGFR = epidermal growth factor receptor.

Pathogenesis of hidradenitis suppurativa



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Hidradenitis Suppurativa

Atopic dermatitis (eczema)	
Pathogenesis	• Mutations in **filaggrin** gene >> skin barrier dysfunction • Type I hypersensitivity reaction. Associated with other atopic diseases
Risk factors	• Low humidity • Relatives with eczema, allergies, or asthma
Clinical features	• **Infant:** Itchy, red, scaly, crusted lesions on extensor surfaces, trunk, cheeks & scalp • **Child/adult**: Lichenified plaques in flexural creases
Treatment	Topical emollients +/- steroid ointment
Complications	• Eczema herpeticum • Cellulitis/abscess • Discomfort interfering with daily activities & sleep

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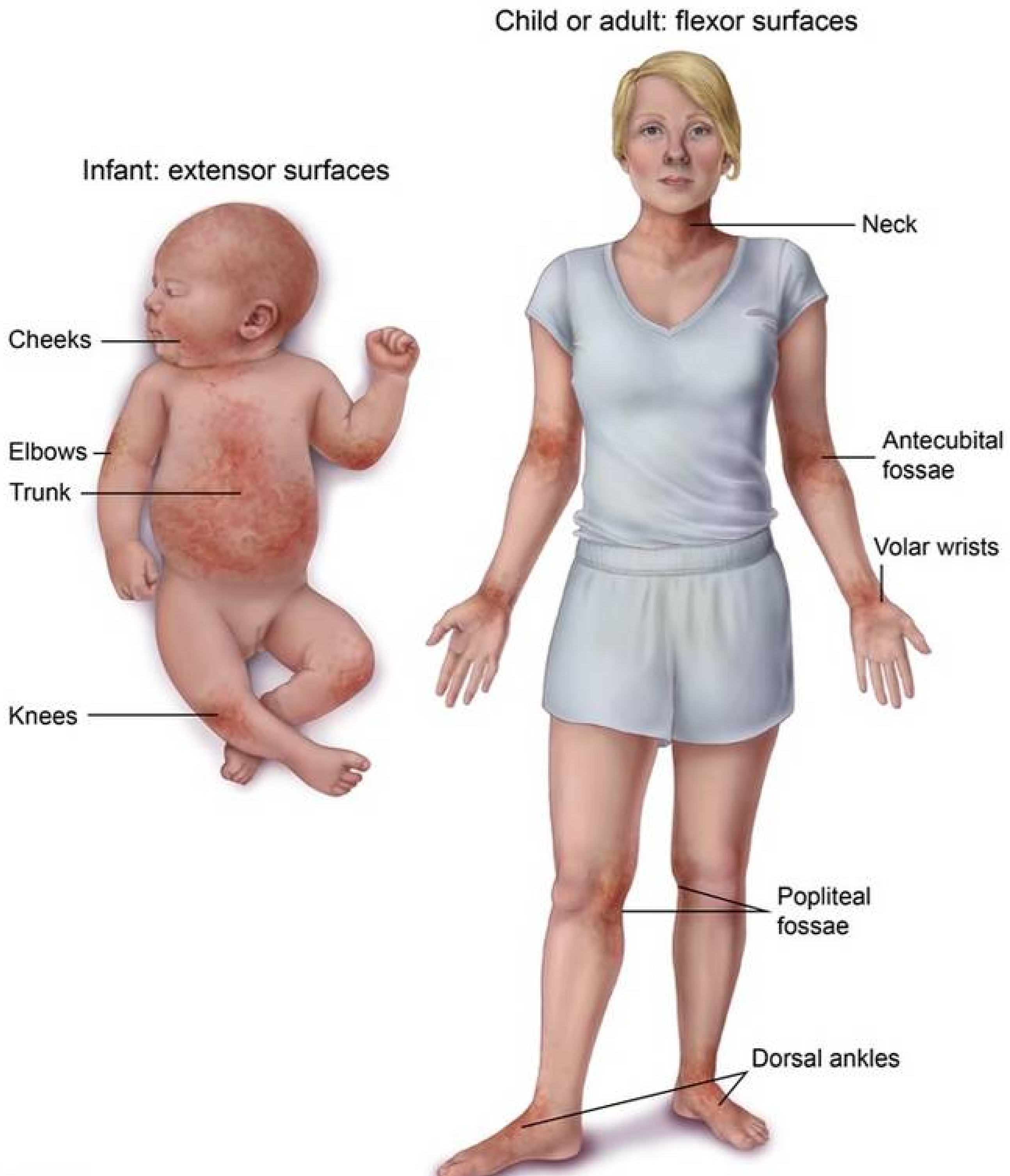
Atopic dermatitis distribution by age



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Eczema>>

lichenification with thickening of Stratum spinosum (acanthosis) and Stratum corneum (hyperkeratosis).

Spongiosis (excessive epidermal extracellular fluid) in acute phase.

Infectious complications of atopic dermatitis		
Diagnosis	**Pathogen**	**Presentation**
Impetigo	*Staphylococcus aureus* *Streptococcus pyogenes*	Painful, non-pruritic pustules with honey-crusted adherent coating
Eczema herpeticum	Herpes simplex type 1	Painful vesicular rash with "punched-out" erosions & hemorrhagic crusting
Molluscum contagiosum	Poxvirus	Flesh-colored papules with central umbilication
Tinea corporis	*Trichophyton rubrum*	Pruritic circular patch with central clearing & raised, scaly border

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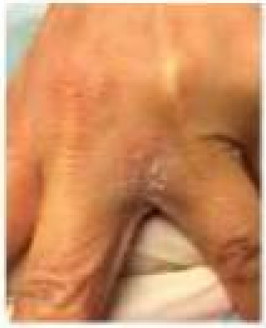
Eczema Herpeticum>>

Ichthyosis vulgaris>>

Inherited disorder of keratinization characterized by generalized scaling of the skin of varying severity.

IV is caused by pathogenic variants in the filaggrin gene (FLG), encoding profilaggrin, a major component of keratohyalin granules in the granular layer of the epidermis.

The disorder manifests in infancy or early childhood as diffuse, dry skin with fine, white or gray scale on the extensor extremities and trunk (picture **B**). Hyperlinear palms (picture **A**)

Contact dermatitis		
	Allergic 	**Irritant** 
Pathophysiology	Type IV hypersensitivity	Physical or chemical irritation
Triggers	• Poison oak/ivy/sumac • Nickel • Rubber/latex • Leather dyes • Medications	• Soaps/detergents • Chemicals • Acid/alkali
Appearance	• Primarily on exposed skin, well demarcated • Erythema • Papules/vesicles • Chronic lichenification	• Commonly on hands • Erythema • Fissures

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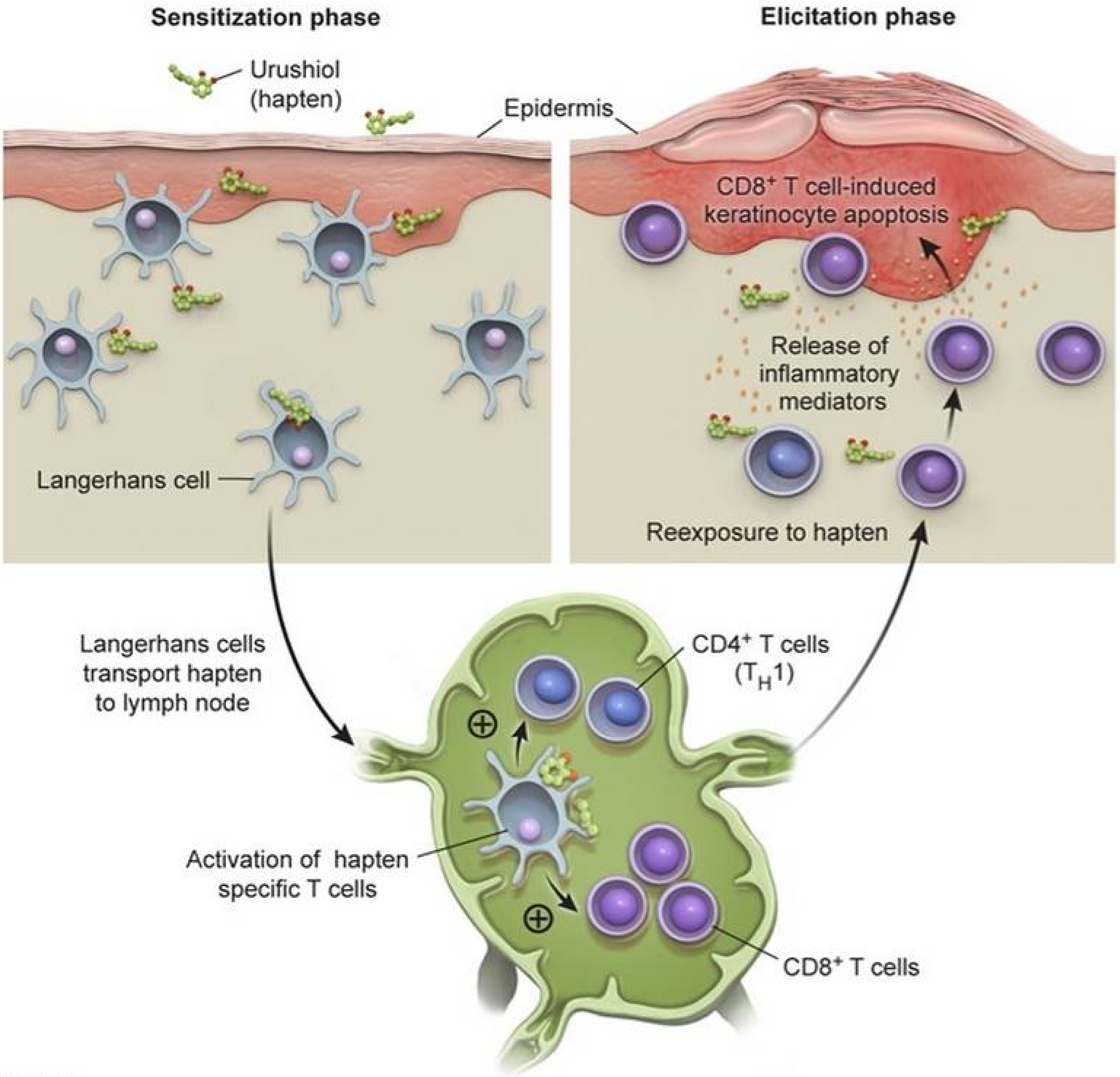
Urushiol-induced contact dermatitis



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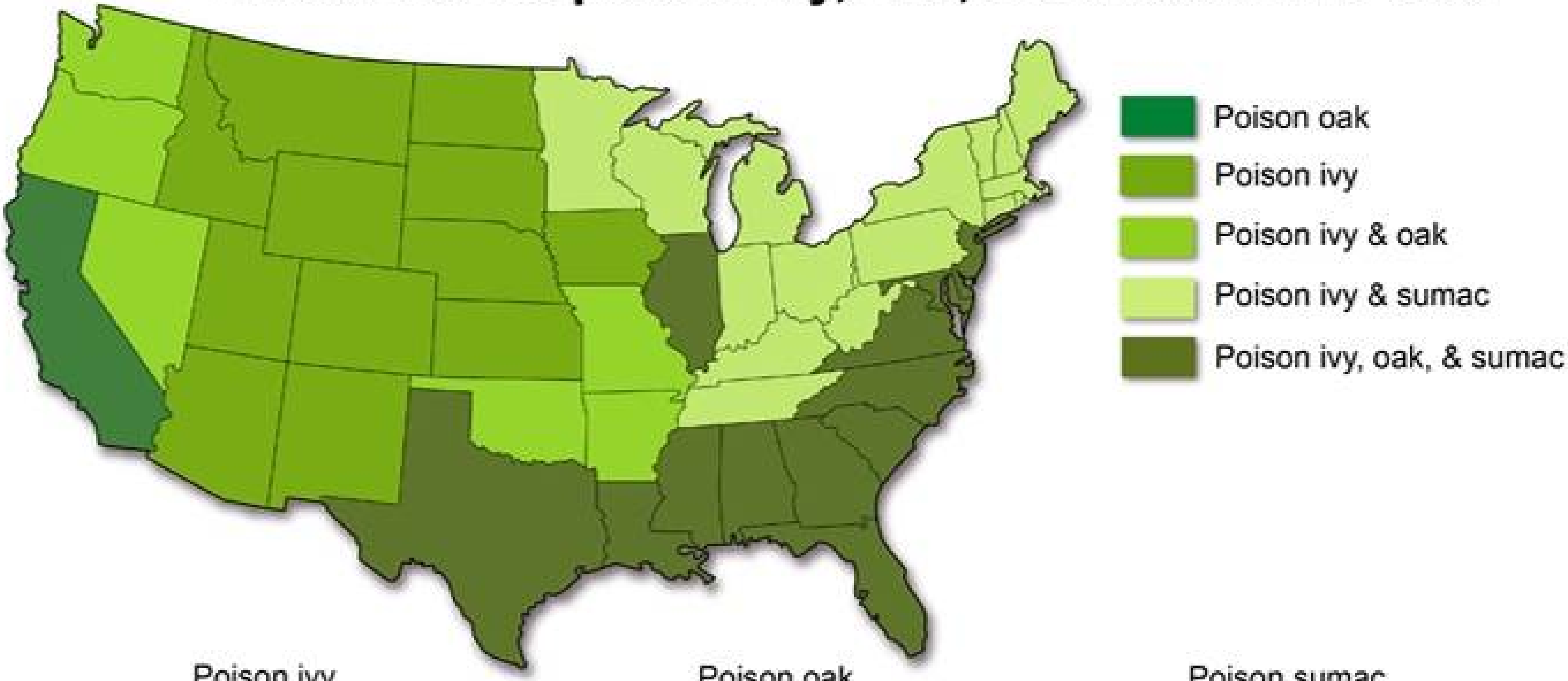
Prevalence of poison ivy, oak, & sumac in the U.S.



Poison ivy

Poison oak

Poison sumac

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Allergic contact dermatitis>>

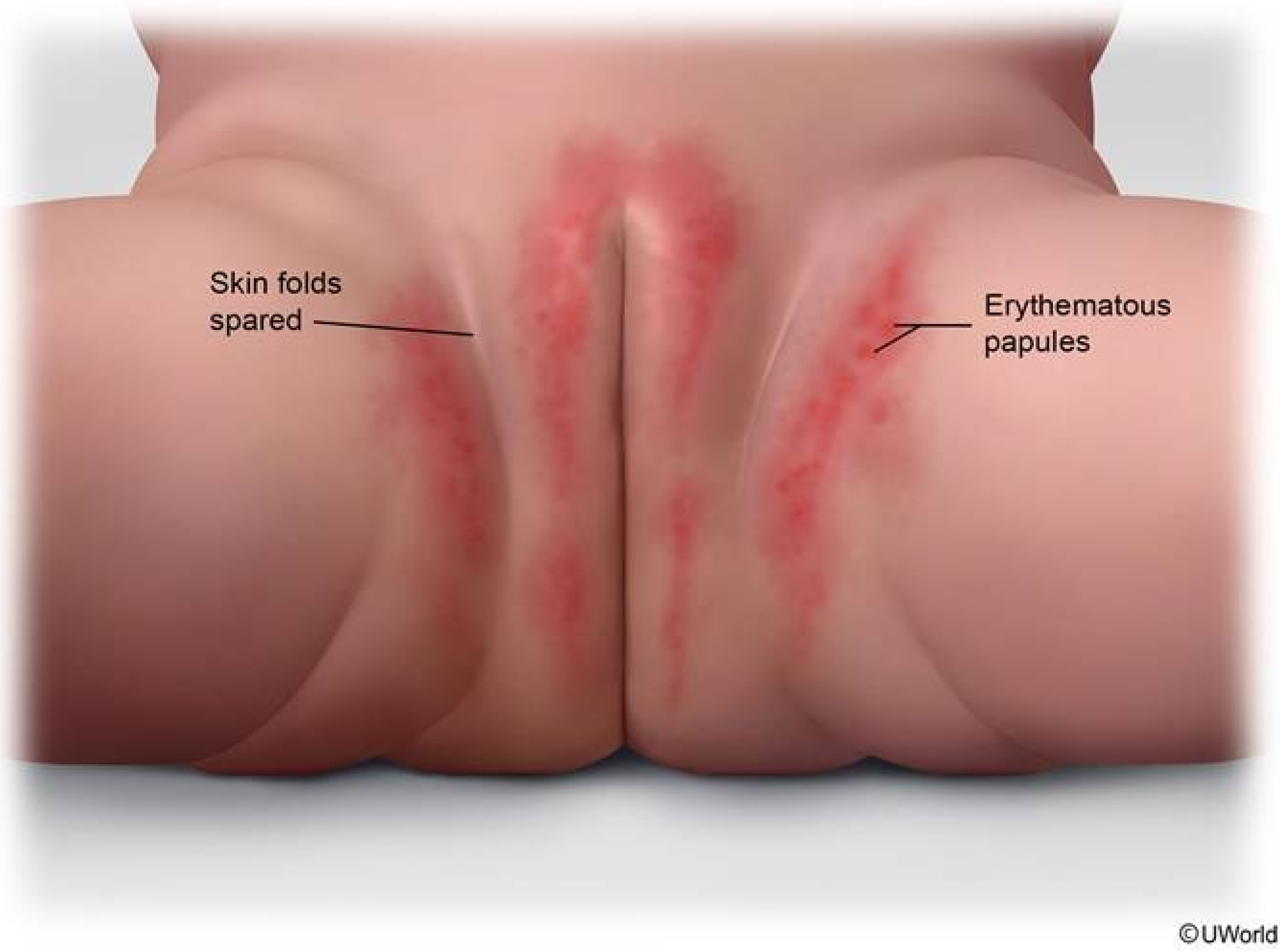
Allergic contact dermatitis>>

Allergic contact dermatitis>>

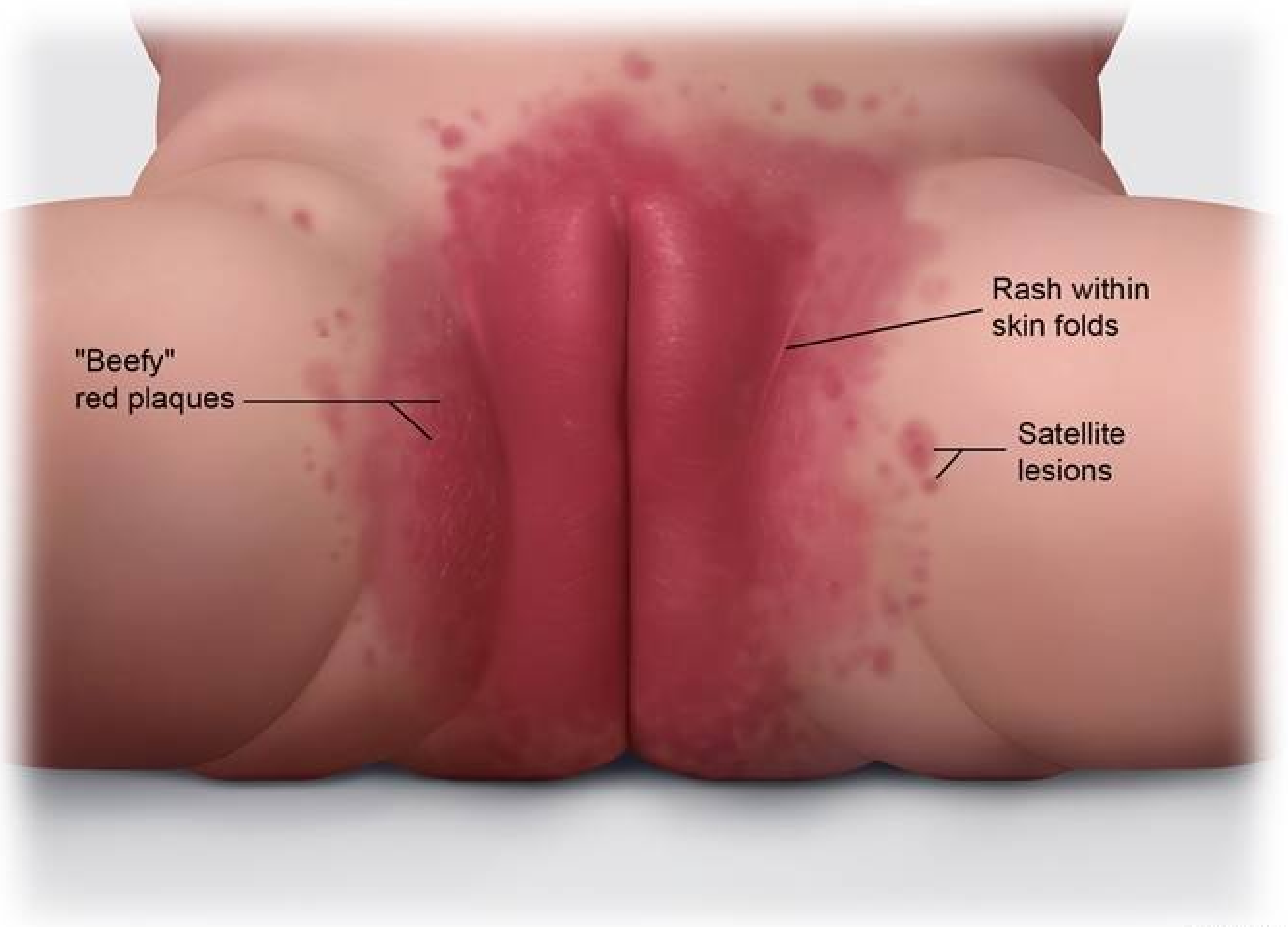
Irritant contact dermatitis>> fissuring and scaling

Irritant contact diaper dermatitis>>
It is the most common diaper rash, characterized by **skin barrier breakdown** as a result of exposure to fecal bacteria that causes **increased local skin pH**. **Treatment** is with a barrier ointment that prevents skin contact with urine and stool.

Irritant diaper dermatitis



Candidal diaper dermatitis



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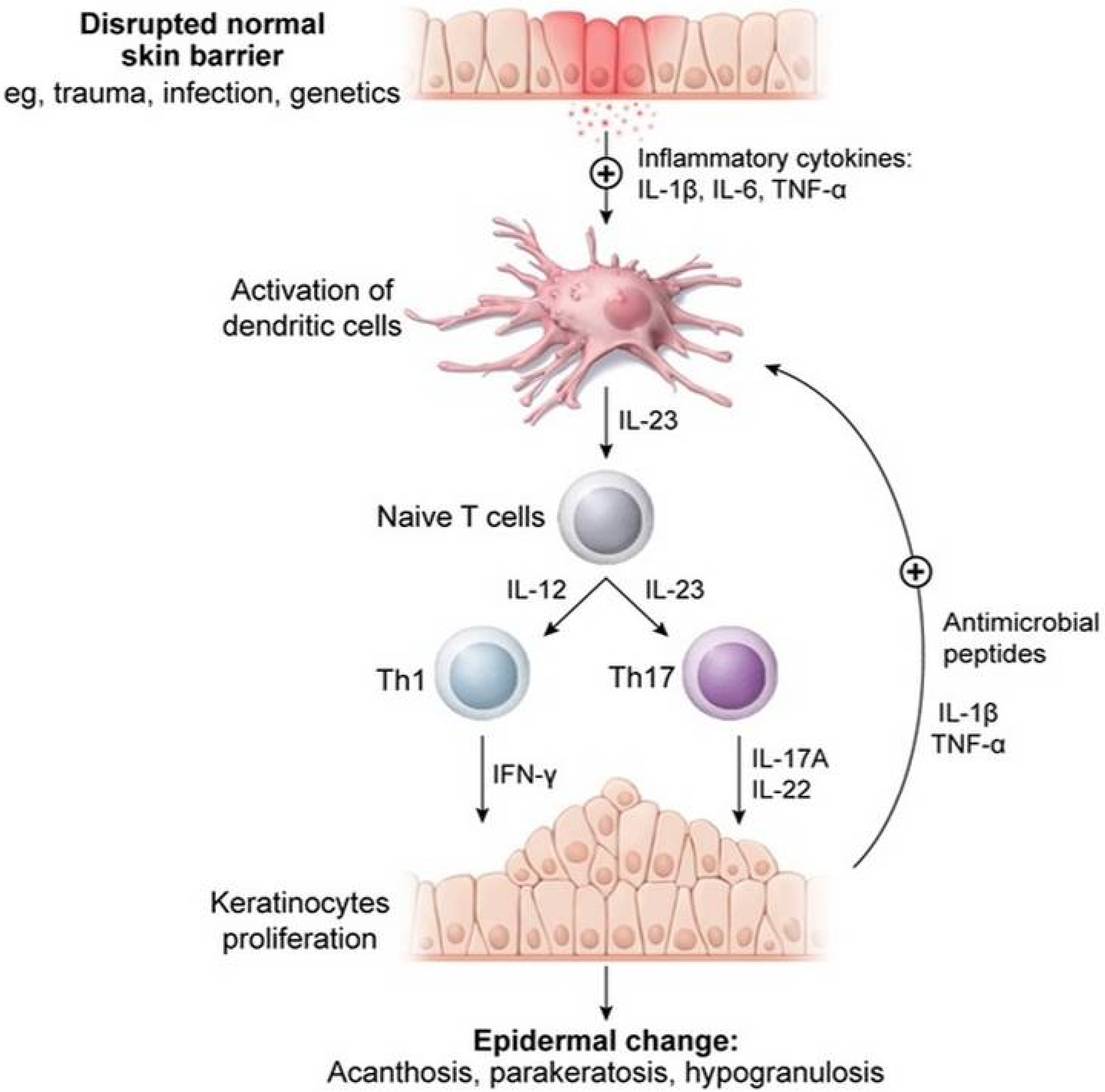
Psoriasis

Papules and plaques with silvery scaling , especially on knees and elbows. Acanthosis with parakeratotic scaling (nuclei still in stratum corneum), Munro microabscesses. ↑ stratum spinosum, ↓ stratum granulosum. Auspitz sign —pinpoint bleeding spots from exposure of dermal papillae when scales are scraped off. Associated with nail pitting and psoriatic arthritis.

Pathophysiology of psoriasis



IFN = interferon; **IL** = interleukin; **TNF** = tumor necrosis factor.

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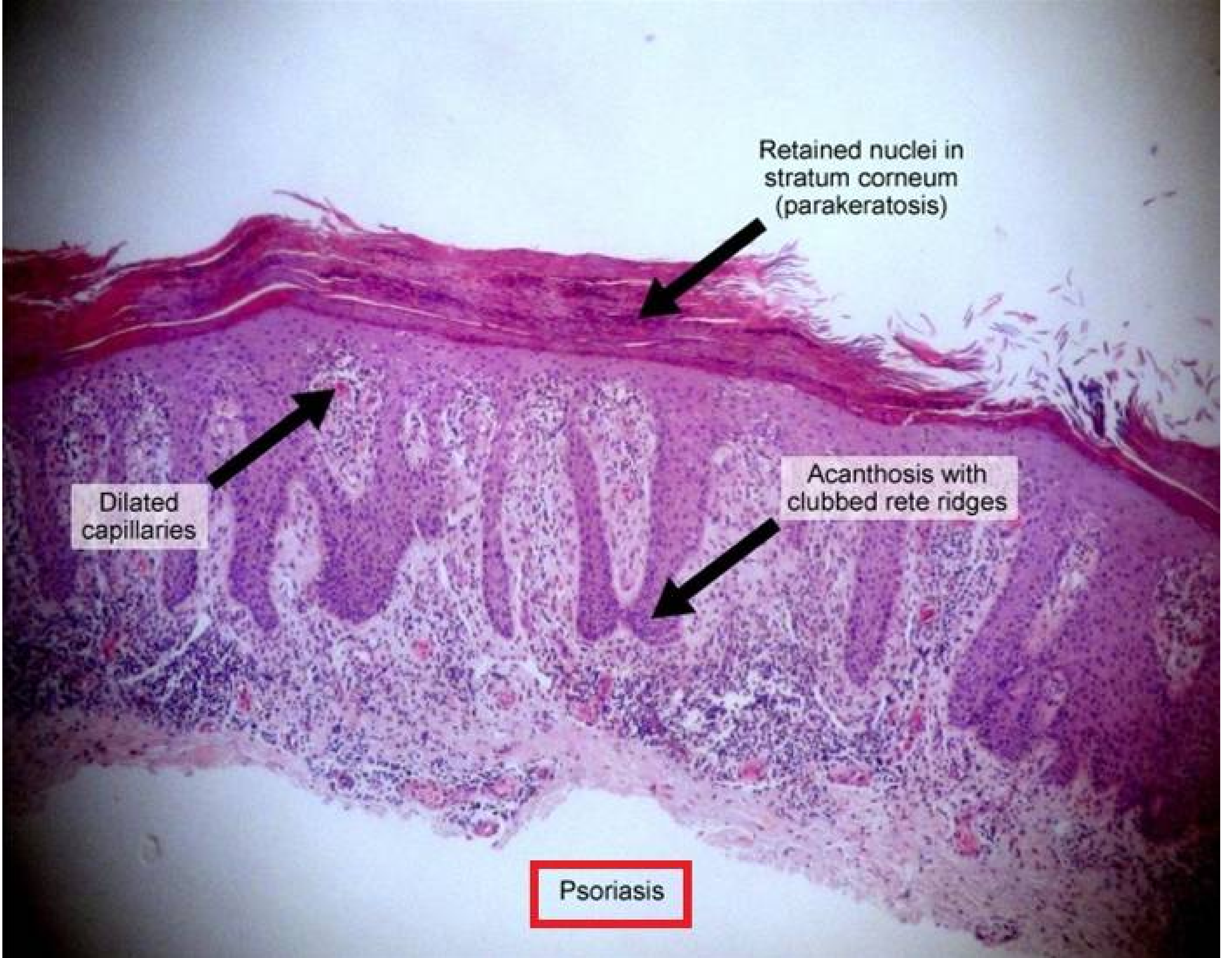

Retained nuclei in stratum corneum (parakeratosis)
Dilated capillaries
Acanthosis with clubbed rete ridges
Psoriasis

Psoriasis> >

erythematous plaques with silvary scales

Plaque psoriasis

Psoriasis

Psoriasis>> polyarthritis resembling RA

Rosacea	
Erythemato-telangiectatic	• Persistent facial erythema/flushing • Telangiectasias
Papulopustular	• Papules & pustules on central face
Ocular	• Conjunctival hyperemia • Lid margin telangiectasias
Treatment	• Avoidance of triggers (eg, alcohol, spicy foods) • Sun protection • Gentle cleansers & emollients • Topical metronidazole for papulopustular type • Laser or topical brimonidine (vasoconstrictive α-2 agonist) for erythematotelangiectatic type

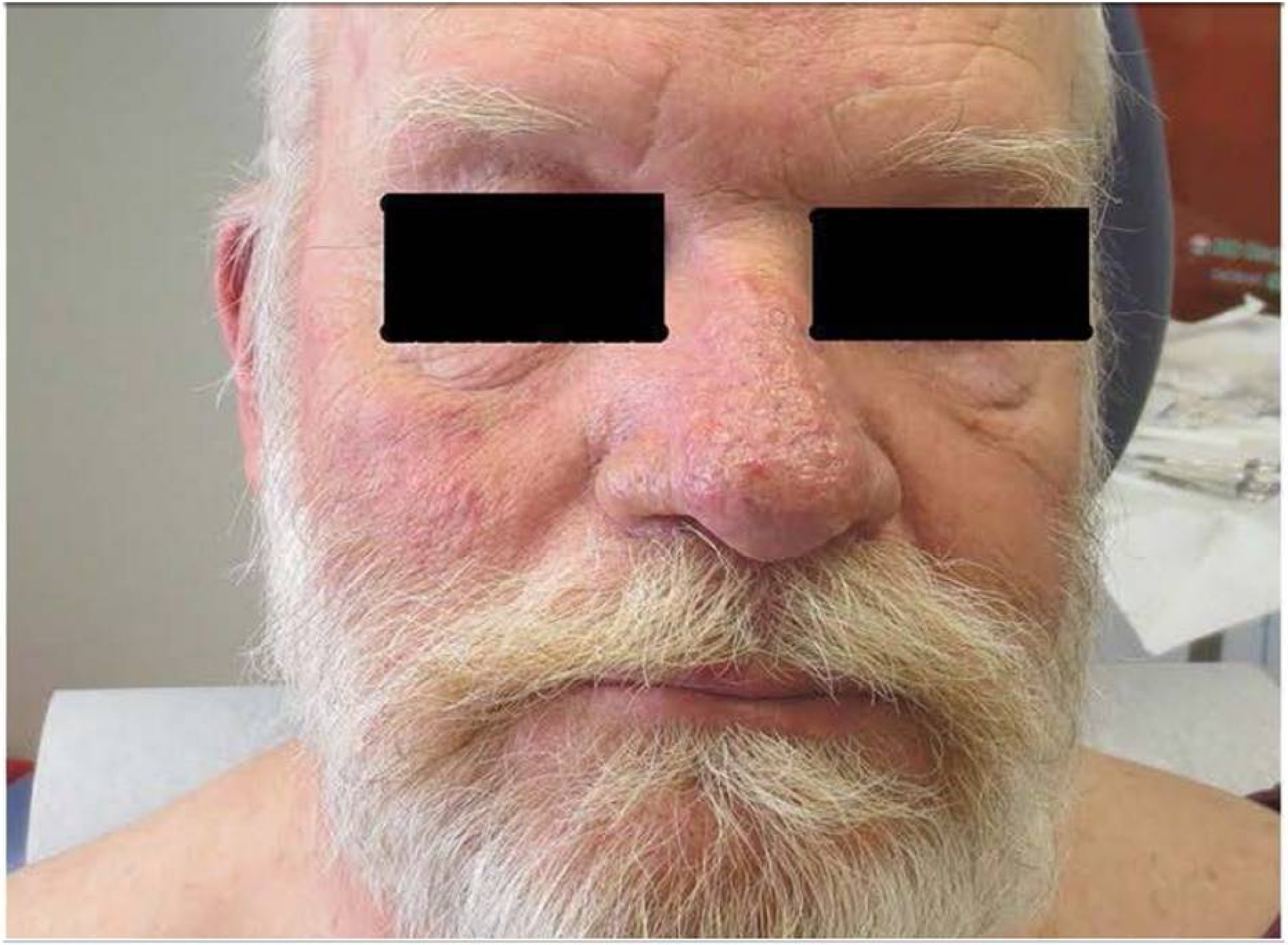
Erythemato-telangiectatic Rosacea

Papulopustular Rosacea

Ocular Rosacea

Phymatous rosacea>>

can cause rhinophyma (bulbous deformation of nose)



Seborrheic keratosis

Flat, greasy, pigmented squamous epithelial proliferation of immature keratinocytes with keratin-filled cysts (horn cysts) . Looks "stuck on." Lesions occur on head, trunk, and extremities. Common benign neoplasm of older persons. Leser-Trélat sign —rapid onset of multiple seborrheic keratoses, indicates possible malignancy (eg, GI adenocarcinoma).

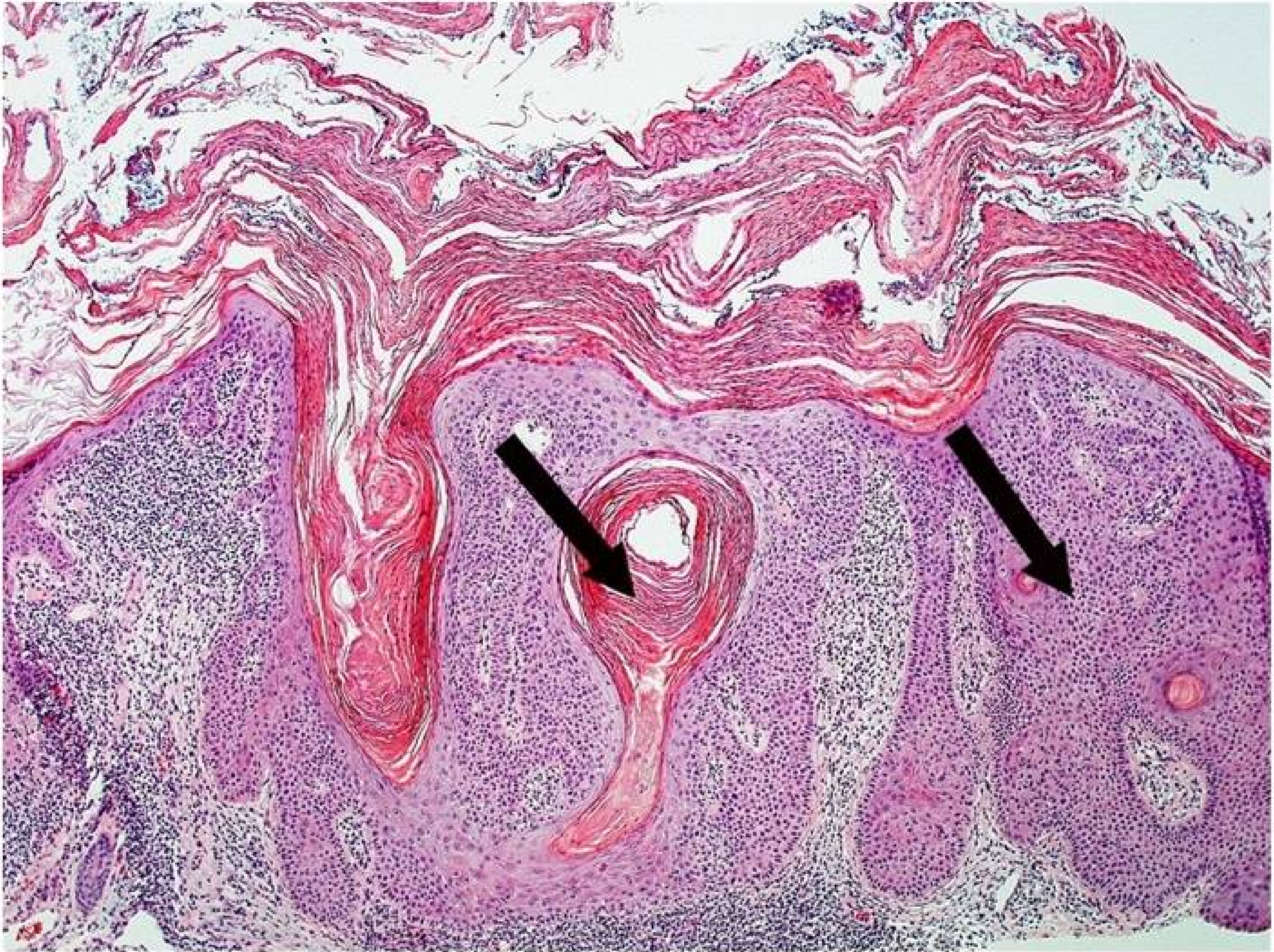
Seborrheic Keratosis

Seborrheic keratosis

Leser-Trelat sign>>

Seborrheic keratosis>>

hyperkeratosis.. keratin-containing cysts



Clinical features of acute urticaria	
Clinical presentation	• Well-circumscribed, raised erythematous plaques • Lesions can be oval, round, or serpiginous up to several centimeters in diameter • Intense pruritus • Lesions can worsen over minutes to hours, then resolve within 24 hours
Etiologies	• Infections (viral, bacterial, parasitic) • IgE mediated (antibiotics, insect bites, latex, food, blood products) • Direct mast cell activation (narcotics, muscle relaxers, radiocontrast medium) • NSAIDs • Idiopathic (up to 50% of patients)

NSAID = nonsteroidal antiinflammatory drug.

Urticaria>> Dermal edema

Angiosarcoma

Rare blood vessel malignancy typically occurring in the head, neck, and breast areas. Usually in elderly, on sun-exposed areas. Associated with radiation therapy and chronic postmastectomy lymphedema. Hepatic angiosarcoma associated with vinyl chloride and arsenic exposures. Very aggressive and difficult to resect due to delay in diagnosis.

Cherry hemangioma

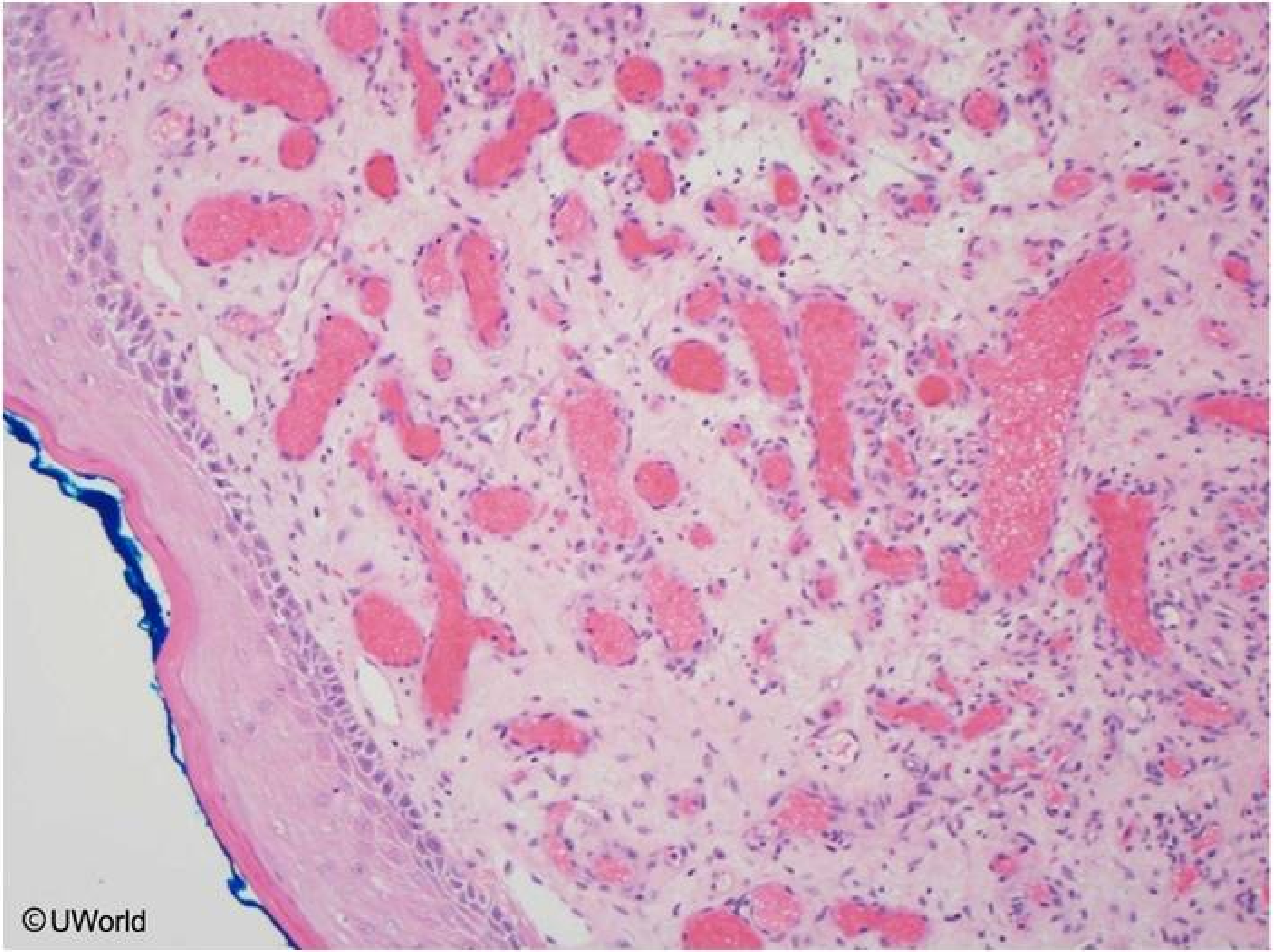
Benign capillary hemangioma commonly appearing in middle-aged adults. Does not regress. Frequency ↑ with age.

Strawberry hemangioma

Benign capillary hemangioma of infancy. Appears in first few weeks of life (1/200 births); grows rapidly and regresses spontaneously by 5–8 years old.

Capillary hemangioma

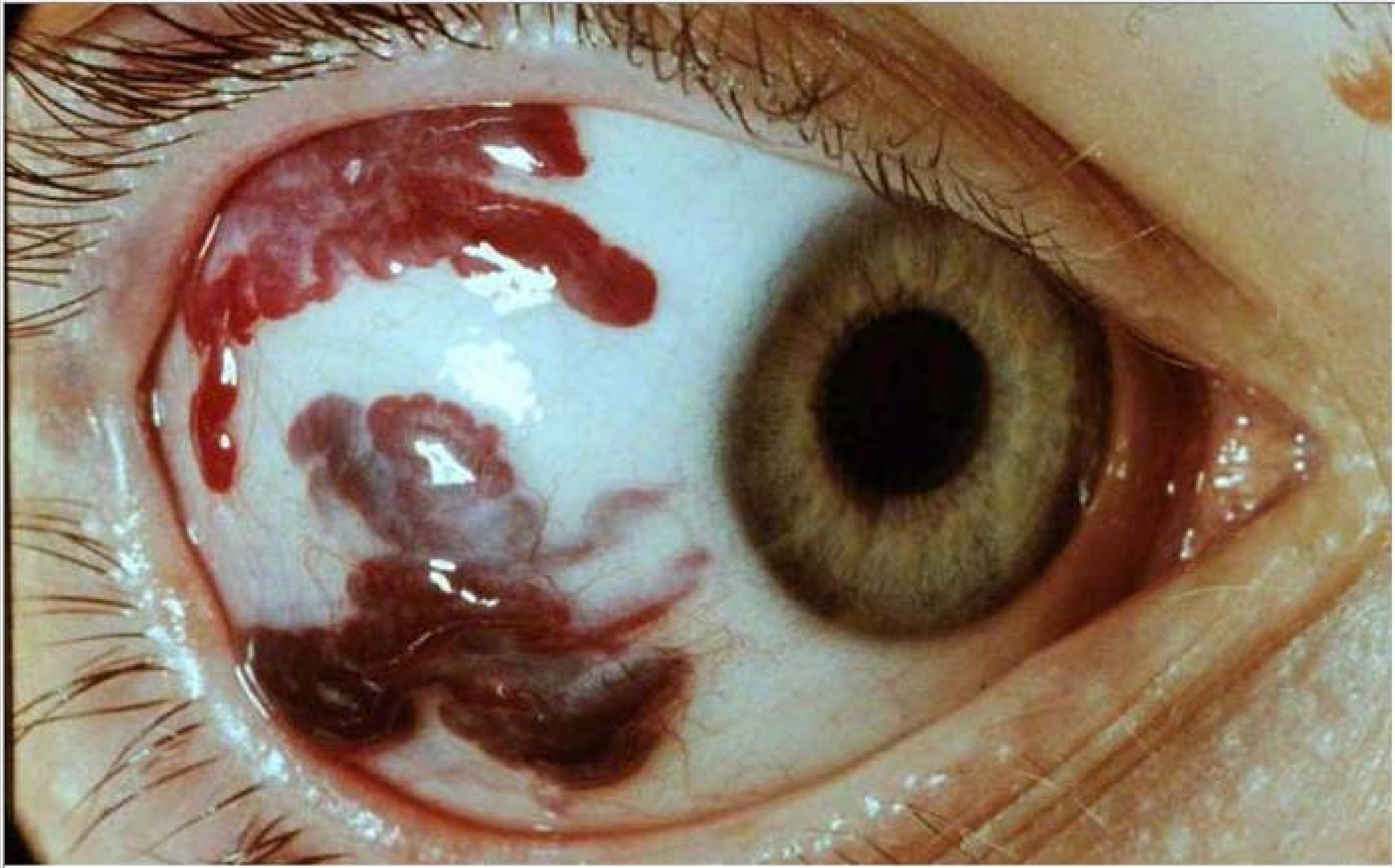
thin-walled blood vessels filled with blood and separated by connective tissue



Pyogenic granuloma>>

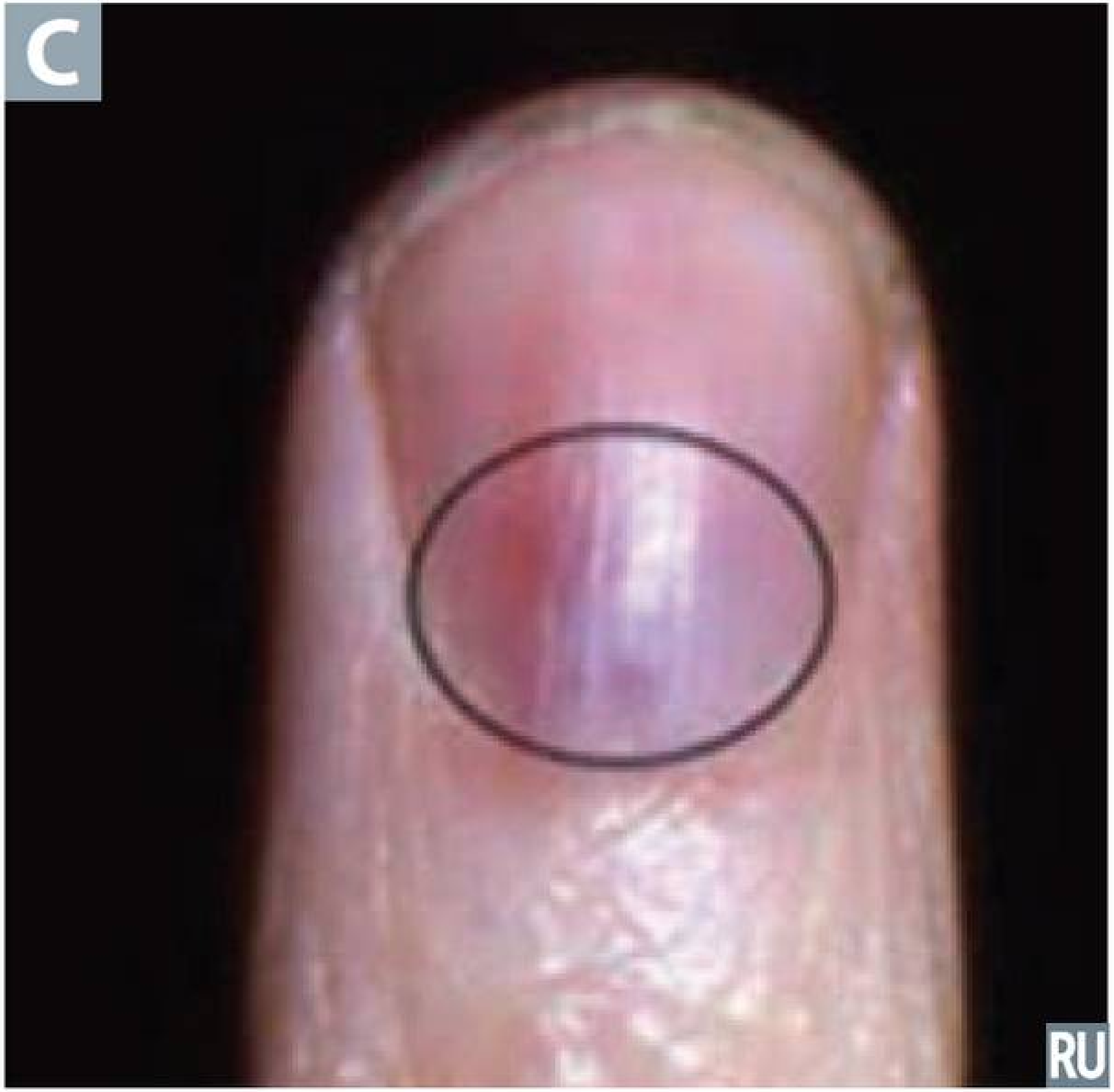
Polypoid lobulated capillary hemangioma that can ulcerate and bleed. Associated with trauma and pregnancy

Hemangioma (Cavernous) of the Conjunctiva



Glomus tumor>>

Benign, painful, red-blue tumor, commonly under fingernails. Arises from modified smooth muscle cells of the thermoregulatory glomus body.



Kaposi sarcoma>>

Endothelial malignancy most commonly affecting the skin, mouth, GI tract, respiratory tract.

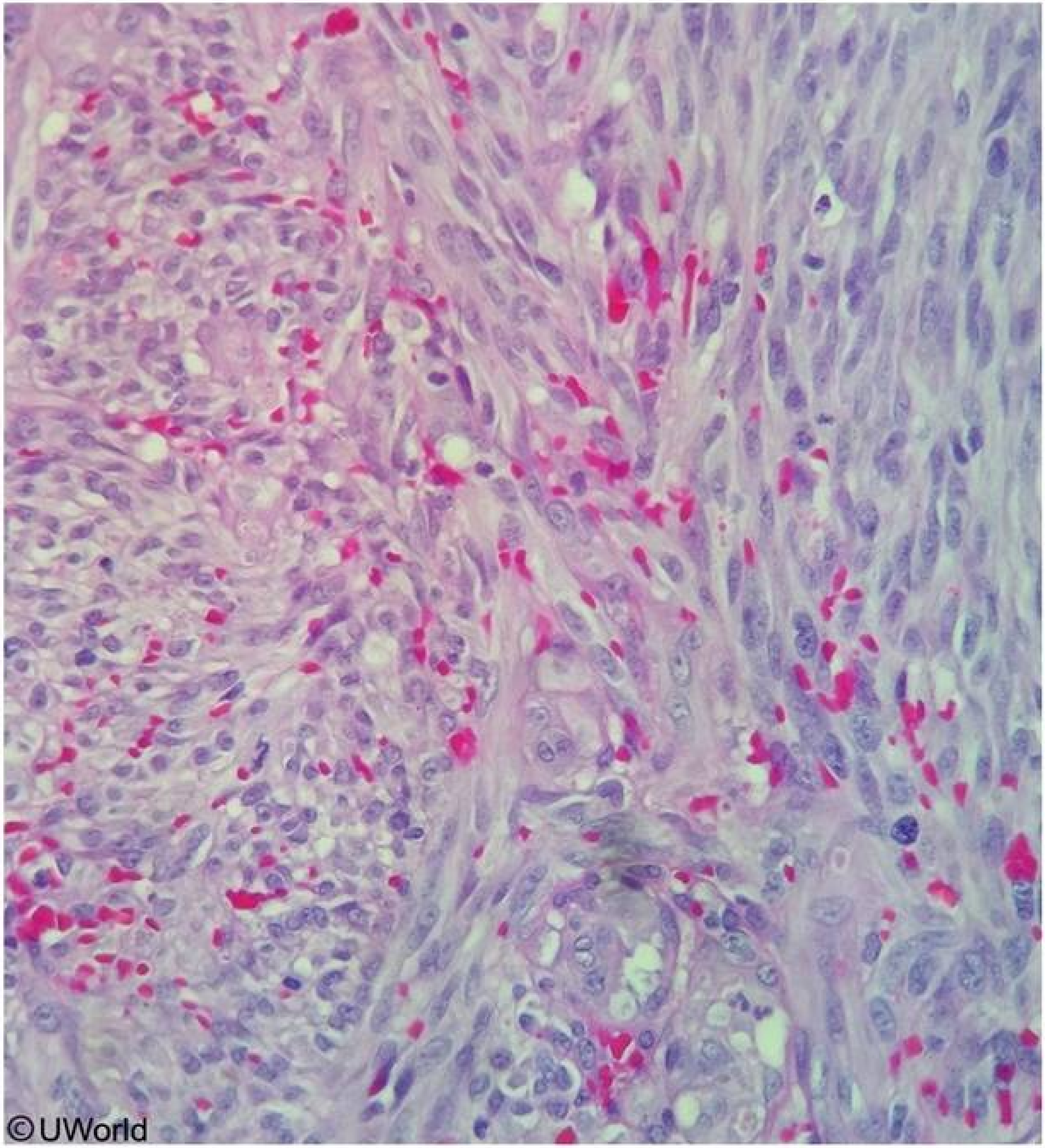
Classically seen in older Eastern European males, patients with **AIDS**, and organ transplant patients. Associated with **HHV-8** and **HIV**.

Rarely mistaken for **bacillary angiomatosis**, but has **lymphocytic infiltrate**.

Kaposi sarcoma

Kaposi sarcoma>>

highly vascular tissue containing elongated spindle cells



Bacillary angiomatosis	
Epidemiology	• ***Bartonella*** *henselae/quintana* • Cat exposure or homelessness (lice) • Severe immunocompromise ○ Advanced **HIV** (CD4 <100/mm^3)
Manifestations	• **Vascular cutaneous lesions** (papular, nodular, peduncular) • **Systemic symptoms** (fever, night sweats, fatigue) • Organ involvement rarely (liver, bone, CNS)
Diagnosis	• Lesional **biopsy** with microscopy/histopathology • Has **neutrophilic infiltrate**
Treatment	• **Doxycycline** or erythromycin • Antiretroviral therapy

Bacillary angiomatosis

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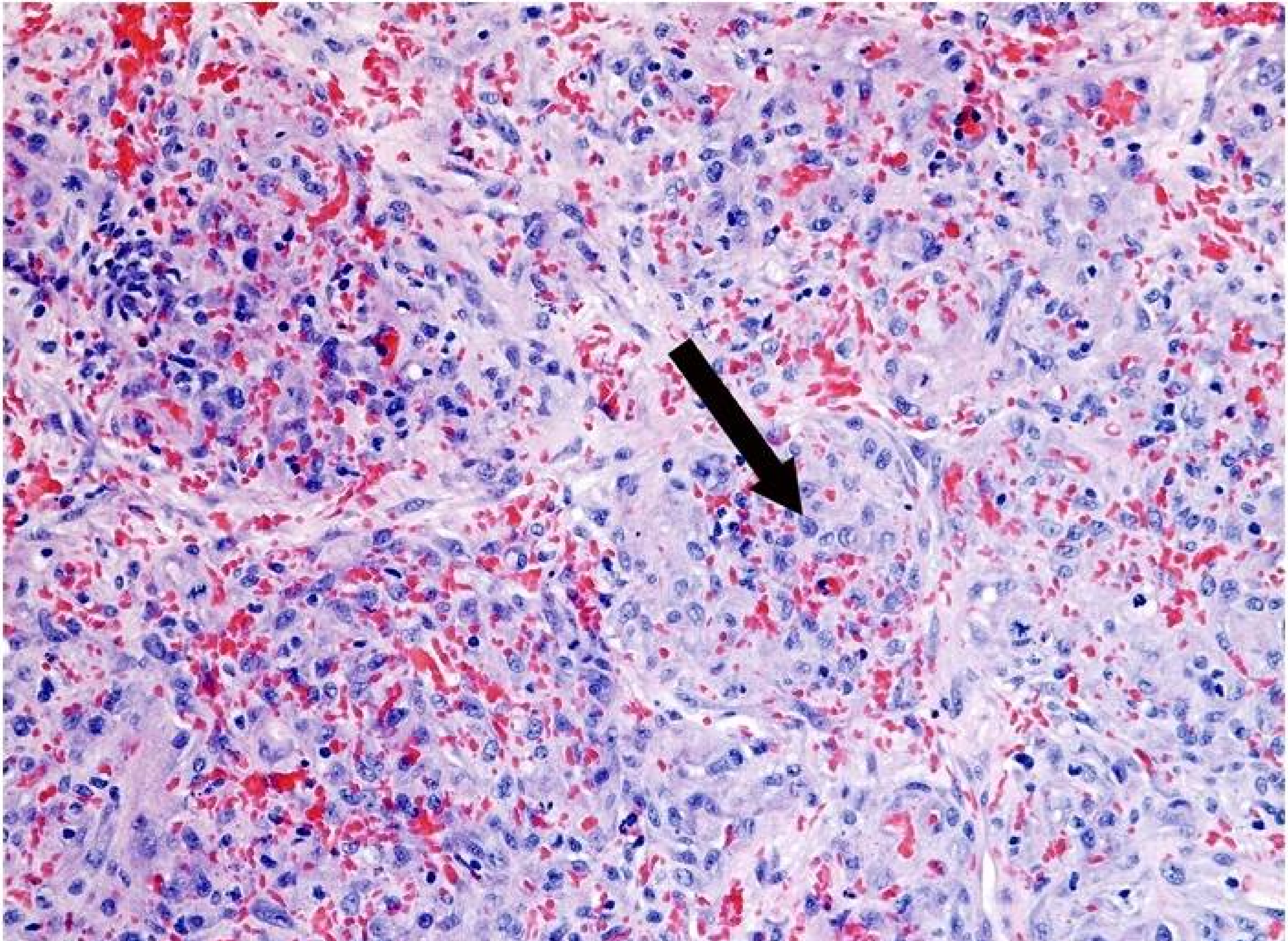
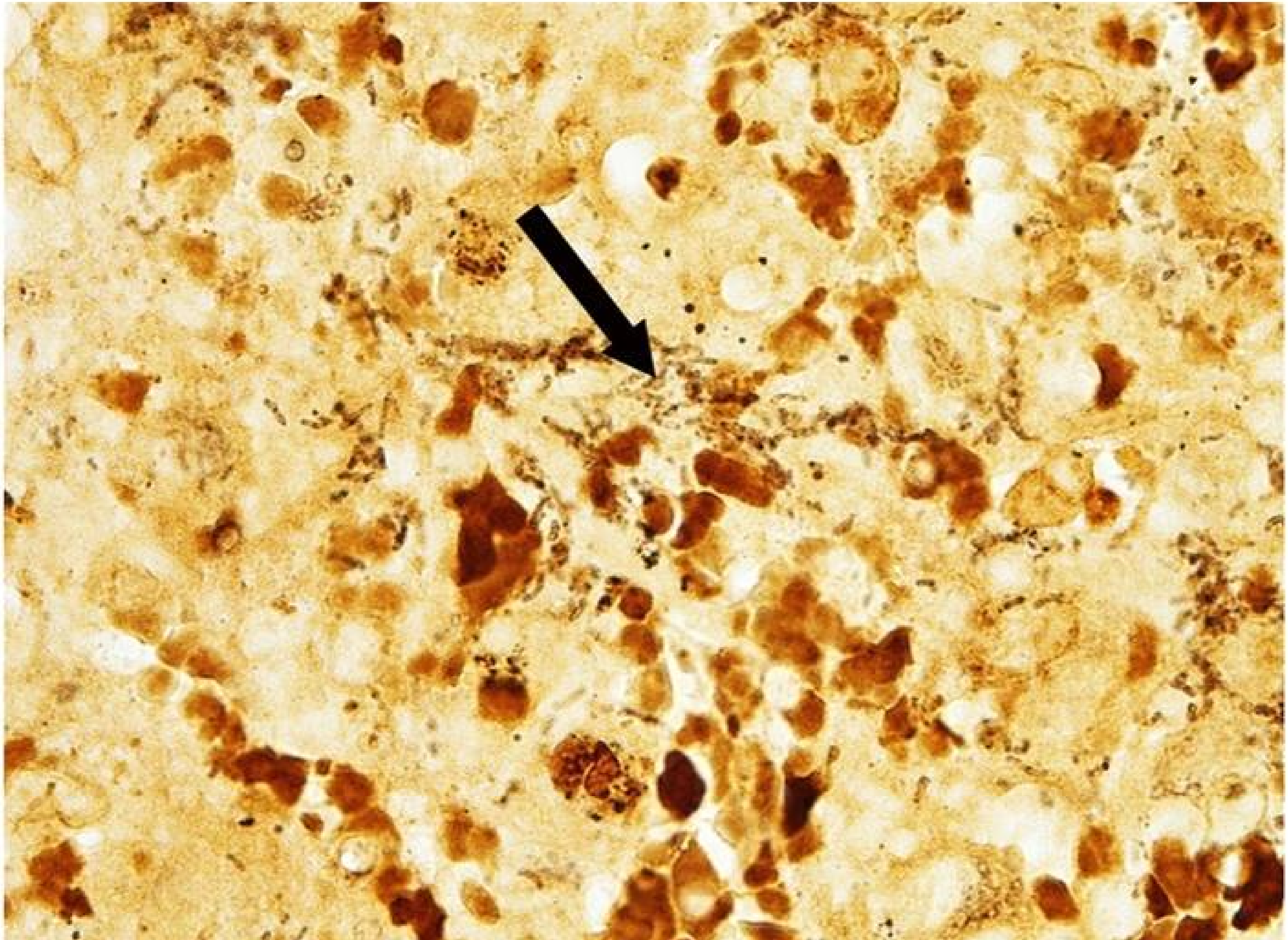
Bacillary angiomatosis>>

large endothelial cells forming vascular channels



Modified silver stain>> Bartonella



Impetigo		
Type	**Non-bullous**	**Bullous**
Microbiology	• *Staphylococcus aureus* • Group *A Streptococcus (S pyogenes)*	• *S aureus*
Clinical features	• Painful non-pruritic pustules • Honey-crusted lesions	• Rapidly enlarging flaccid bullae with yellow fluid • Collarette of scale at periphery of ruptured lesions
Treatment	• Limited skin involvement: Topical antibiotics (eg, mupirocin) • Extensive skin involvement: Oral antibiotics (eg, cephalexin, dicloxacillin, clindamycin)	
Complications	• Poststreptococcal glomerulonephritis	

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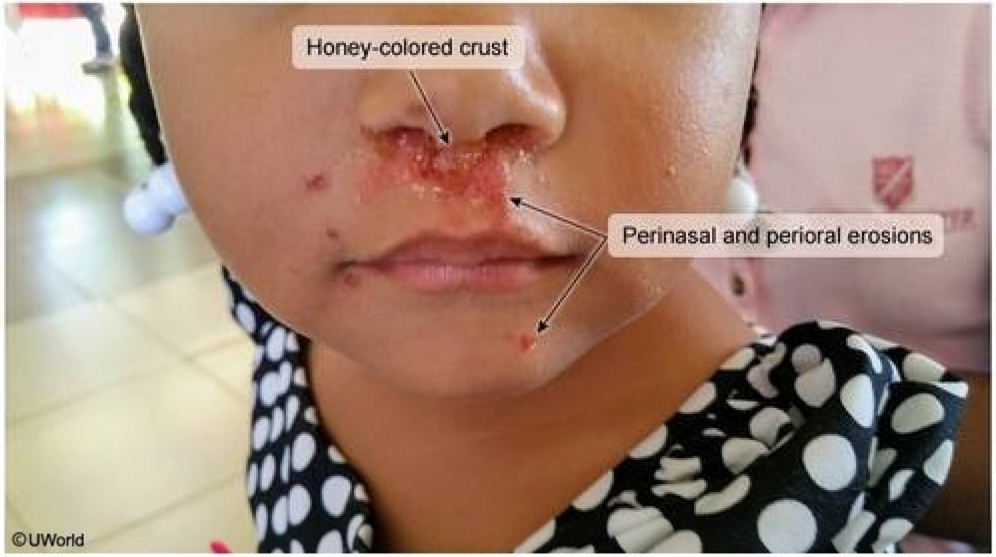
Impetigo>>

Honey colored crusting



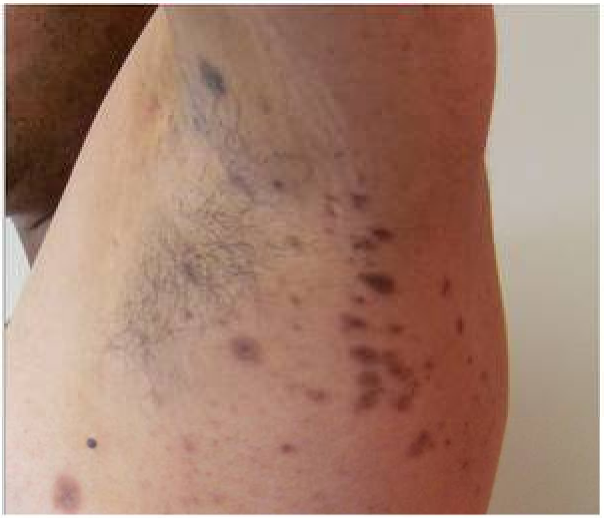
Common skin infections		
Infection	**Organism**	**Manifestations**
Erysipelas	• *Streptococcus pyogenes*	• **Superficial** dermis & lymphatics • Raised, sharply **demarcated edges** • **Rapid** spread & onset • **Fever** early in course
Cellulitis (nonpurulent)	• *S pyogenes* • MSSA	• **Deep** dermis & subcutaneous fat • **Flat edges** with poor demarcation • **Indolent** (over days) • Localized (fever later in course)
Cellulitis (purulent)	• MSSA • MRSA	• Purulent drainage • Folliculitis: Infected hair follicle • Furuncles: Folliculitis → dermis → abscess • Carbuncle: Multiple furuncles

Erysipelas>>

Nonpurulent cellulitis>>

Folliculitis>>

Furuncles>>



Carbuncle

Features of necrotizing fasciitis	
Microbiology	• *Streptococcus pyogenes* (group A streptococci) • *Staphylococcus aureus* • *Clostridium perfringens* • Polymicrobial
Pathogenesis	• Bacteria spread rapidly through subcutaneous tissue & deep fascia, undermining the skin • Most commonly involves extremities & perineal region
Clinical manifestations	• Often antecedent history of minor trauma • Erythema of overlying skin • Swelling & edema • Pain **out of proportion** to examination findings • Systemic symptoms (eg, **fever & hypotension**)
Treatment	• Requires **surgical debridement** & broad-spectrum antibiotics

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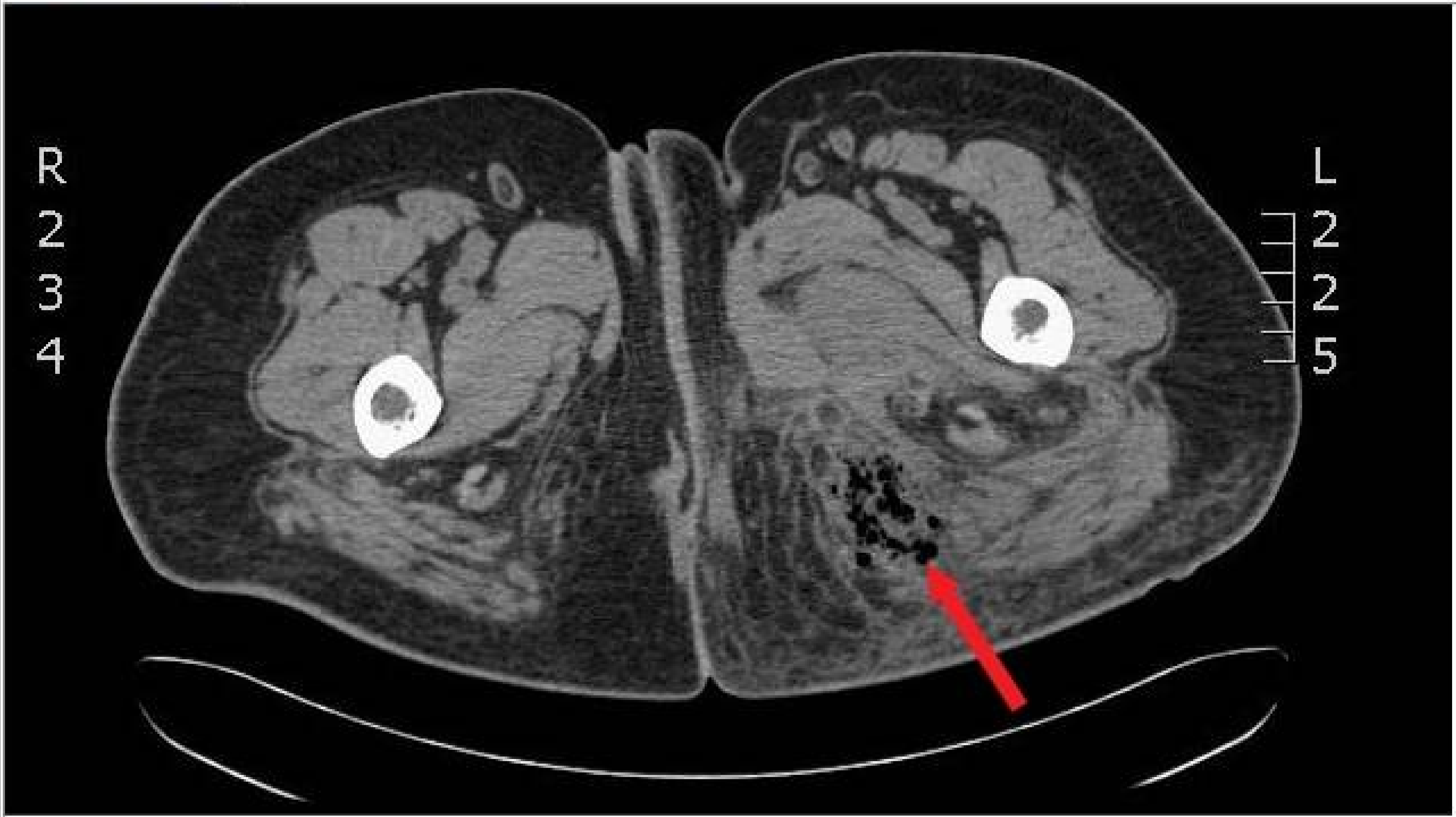
Necrotizing Fasciitis



Staphylococcal scalded skin syndrome

Exotoxin destroys keratinocyte attachments in stratum granulosum only (vs toxic epidermal necrolysis, which destroys epidermal-dermal junction). Characterized by fever and generalized erythematous rash with sloughing of the upper layers of the epidermis G that heals completely. ⊕ Nikolsky sign (separation of epidermis upon manual stroking of skin). Commonly seen in newborns and children/adults with renal insufficiency.

Herpes simplex virus-1>>

ROUTE OF TRANSMISSION	CLINICAL SIGNIFICANCE	NOTES
Respiratory secretions, saliva	Gingivostomatitis, keratoconjunctivitis , herpes labialis (cold sores) , herpetic whitlow on finger, temporal lobe encephalitis, esophagitis, erythema multiforme	Most commonly latent in trigeminal ganglia Most common cause of sporadic encephalitis, can present as altered mental status, seizures, and/or aphasia

Herpangina versus herpetic gingivostomatitis		
Diagnosis	**Herpangina**	**Herpetic gingivostomatitis**
Etiology	Coxsackie A virus	Herpes simplex virus type 1
Age	3-10 years	6 months-5 years
Seasonality	Summer/early fall	None
Clinical features	• Fever • Pharyngitis • Gray vesicles/ulcers on posterior oropharynx	• Fever • Pharyngitis • Erythematous gingiva • Clusters of small vesicles on anterior oropharynx
Treatment	Supportive management	Oral acyclovir

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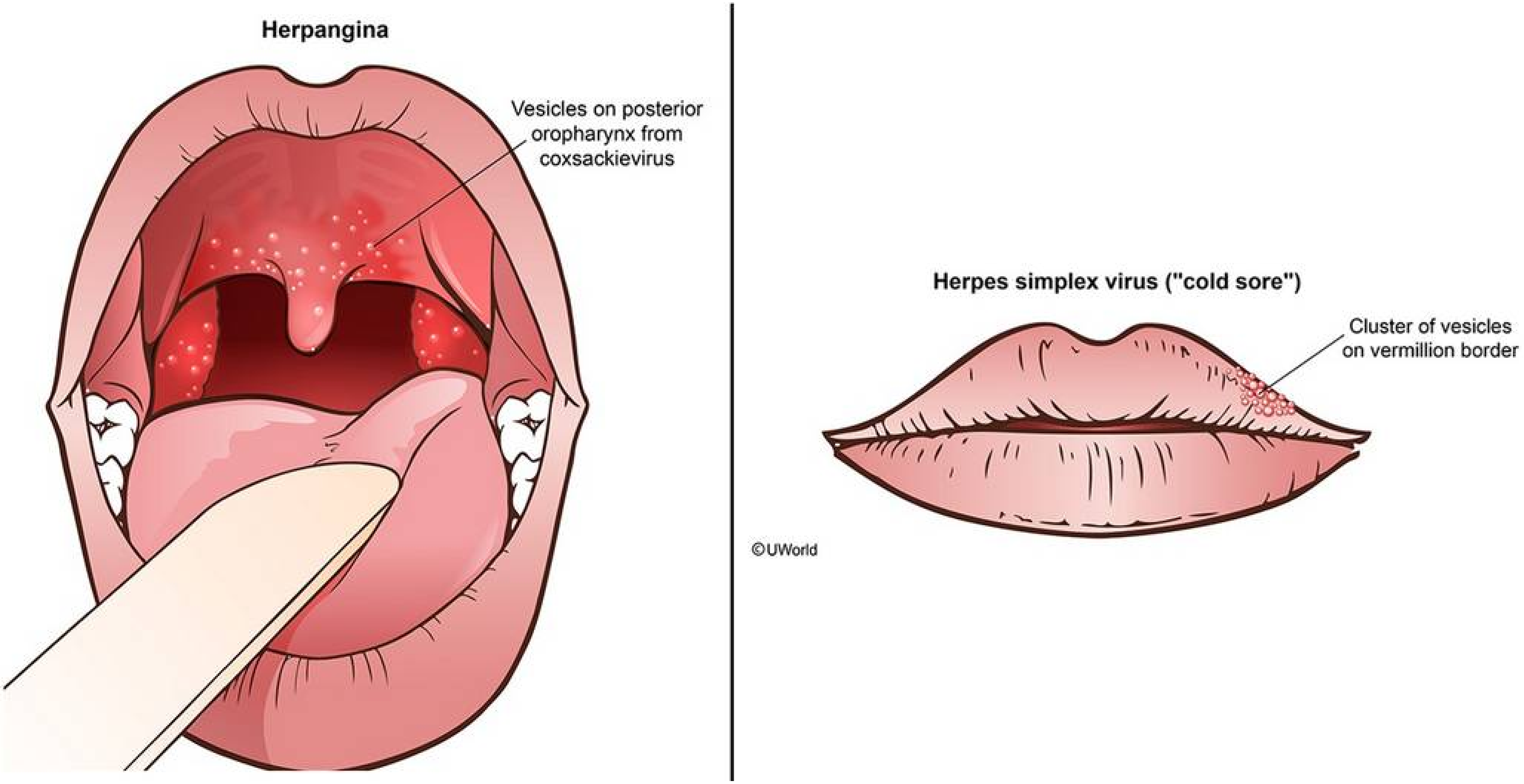
Herpangina
Vesicles on posterior oropharynx from coxsackievirus
Herpes simplex virus ("cold sore")
Cluster of vesicles on vermillion border
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Recurrent herpes>> papules and vesicles

HSV infection> >

Vesicles on erythematous base… ulcers then crusting

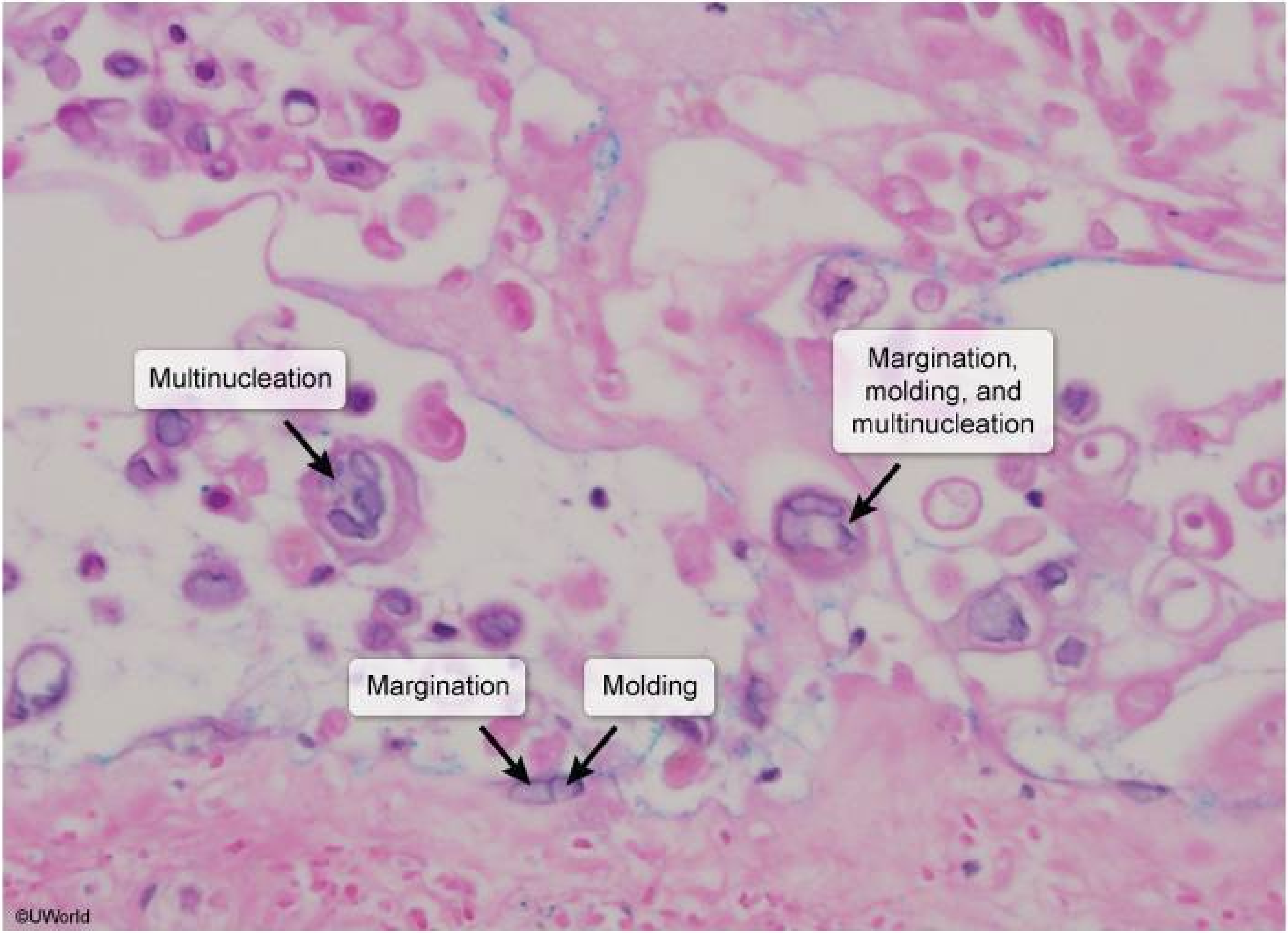
Herpetic whitlow>>

Herpes simplex virus-2>>

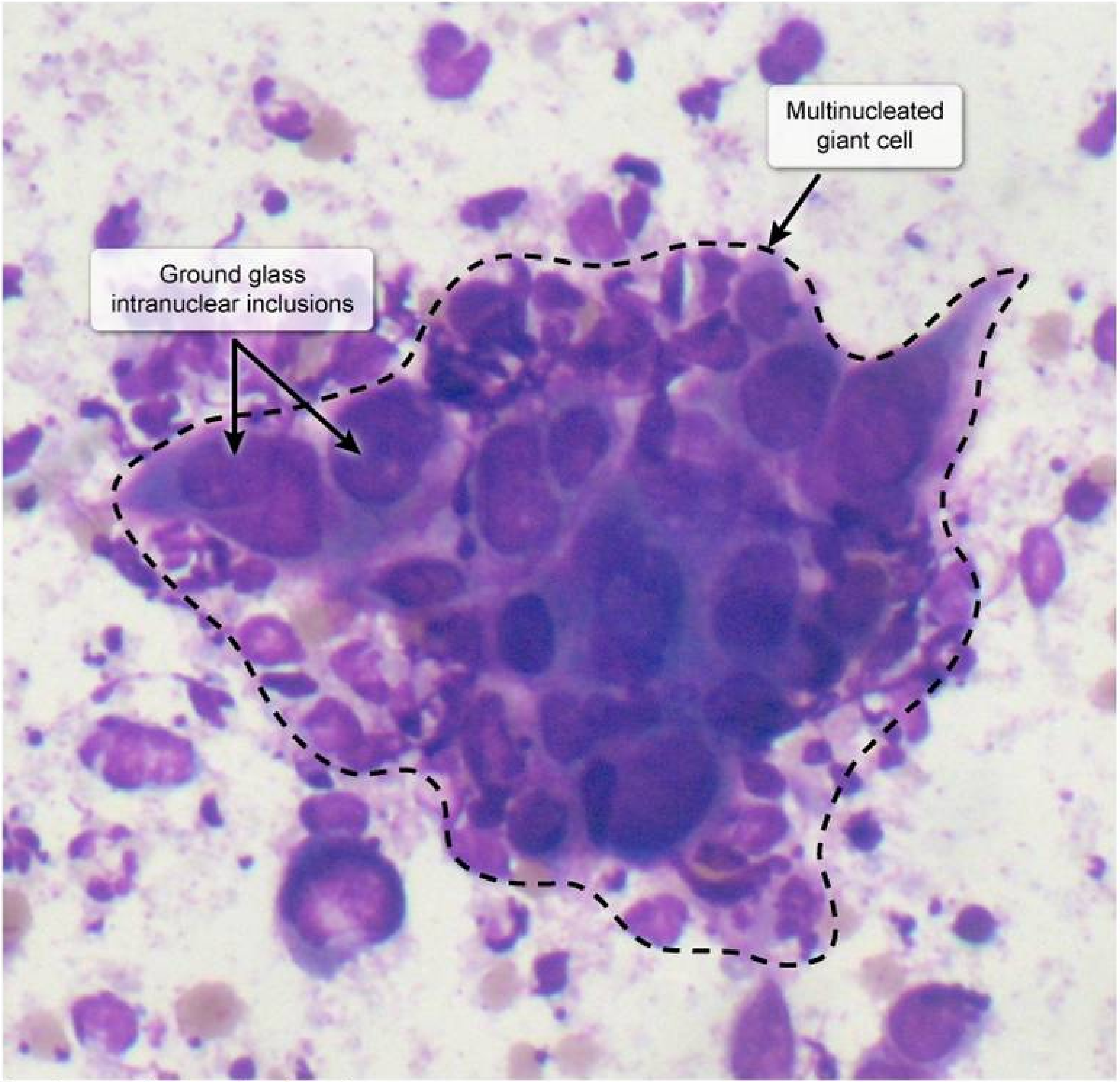
ROUTE OF TRANSMISSION	CLINICAL SIGNIFICANCE	NOTES
Sexual contact, perinatal	Herpes genitalis, neonatal herpes	Most commonly latent in sacral ganglia Viral meningitis more common with HSV-2 than with HSV-1

Genital herpes	
Etiology	HSV-2 (most common)
Clinical presentation	• Cluster of painful vesicles, pustules &/or ulcers on labia, penis, buttocks, or thigh • ± Systemic symptoms in primary infection
Treatment	Acyclovir, famciclovir, or valacyclovir
HSV = herpes simplex virus.	

Tzanck smear>> HSV infection



Herpes simplex



Tzanck preparation – scraping of ulcer base

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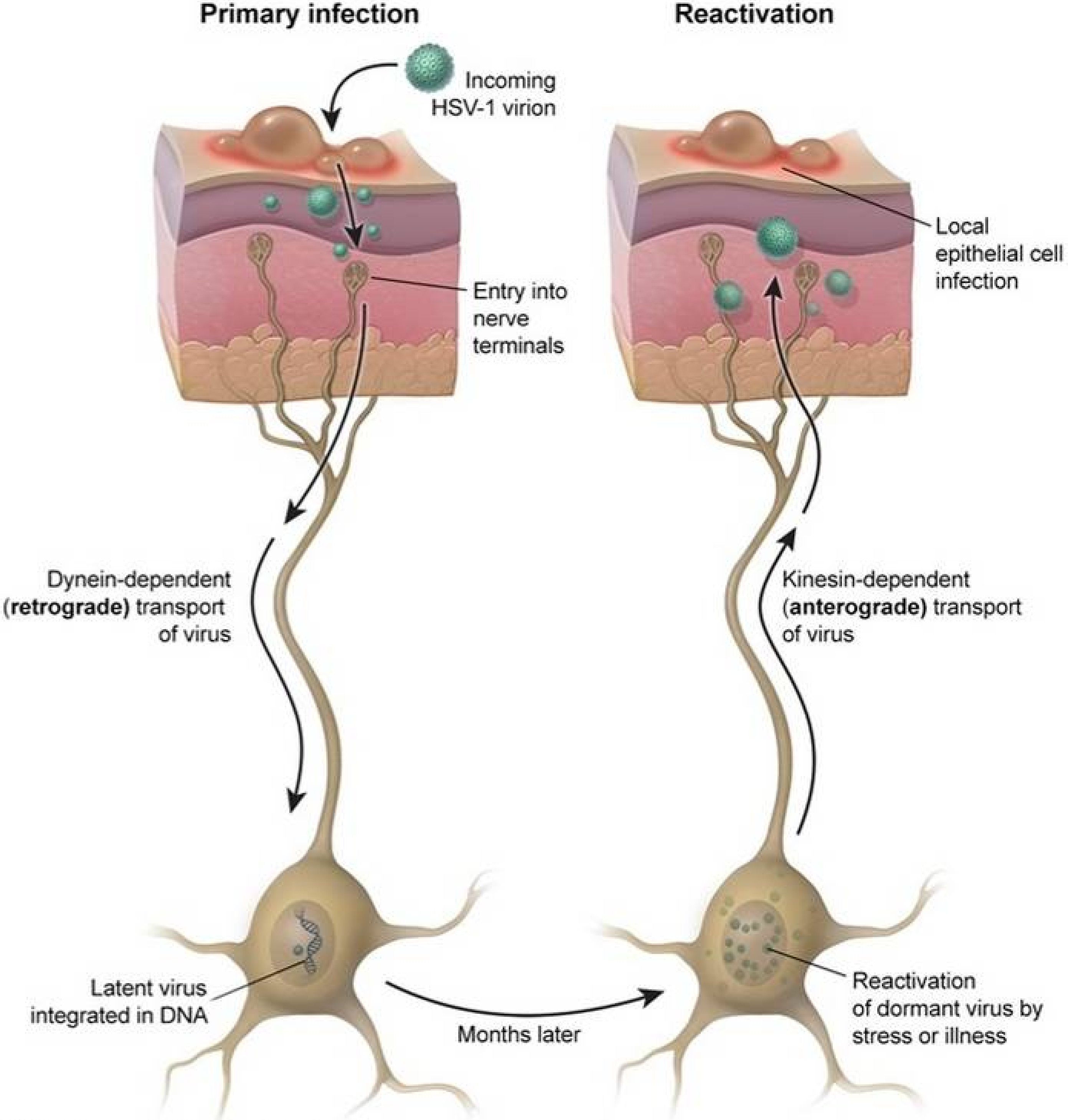
Herpes simplex virus



HSV-1 = herpes simplex virus type 1.

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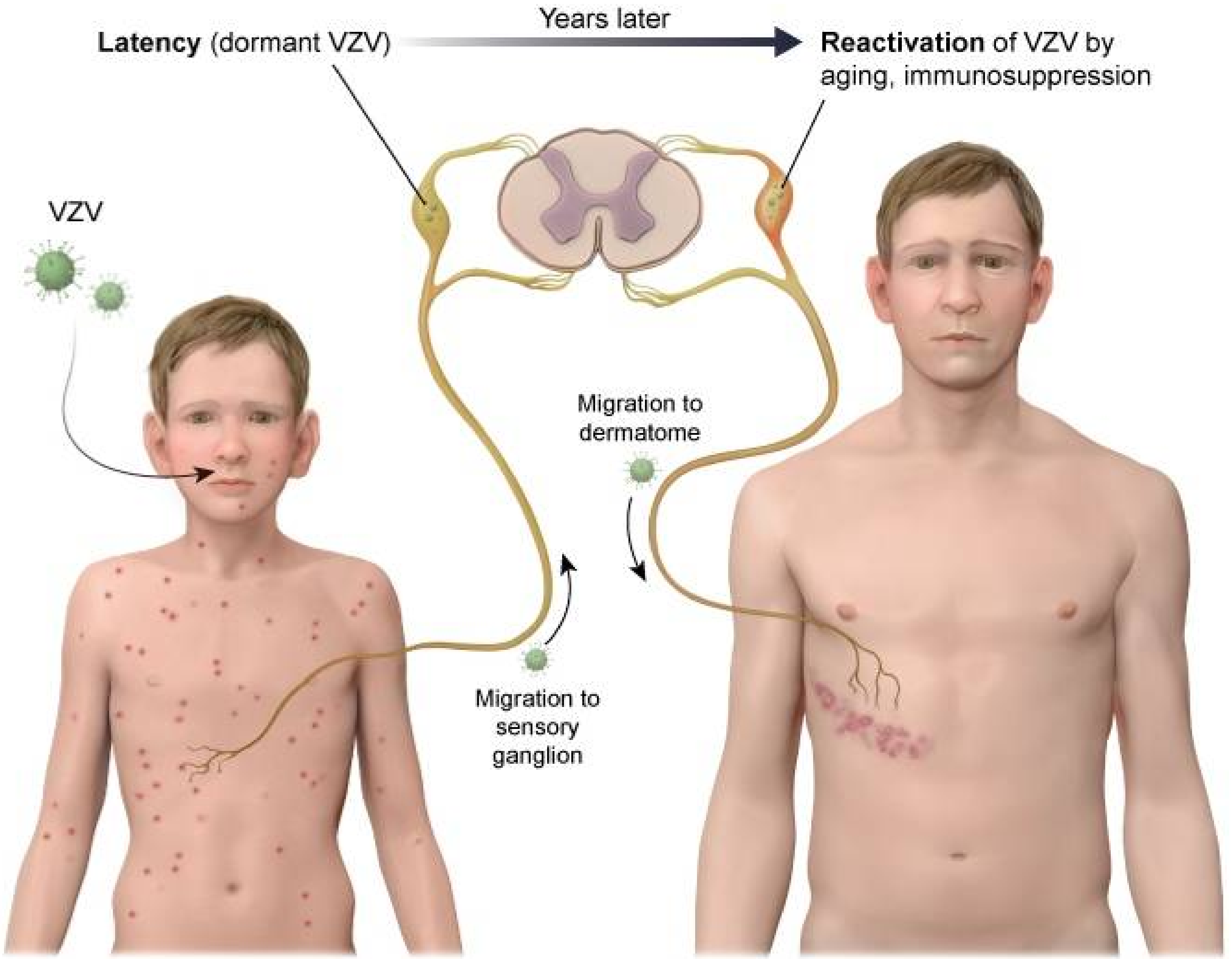
Varicella-Zoster virus (HHV-3)>>

ROUTE OF TRANSMISSION	CLINICAL SIGNIFICANCE	NOTES
Respiratory secretions, contact with fluid from vesicles	Varicella-zoster (chickenpox, shingles), encephalitis, pneumonia Most common complication of shingles is post-herpetic neuralgia	Latent in dorsal root or trigeminal ganglia; CN V_1 branch involvement can cause herpes zoster ophthalmicus

Varicella-zoster virus (VZV) infection



Varicella (chickenpox)
Generalized vesicular rash

Herpes zoster (shingles)
Unilateral, linear vesicular rash

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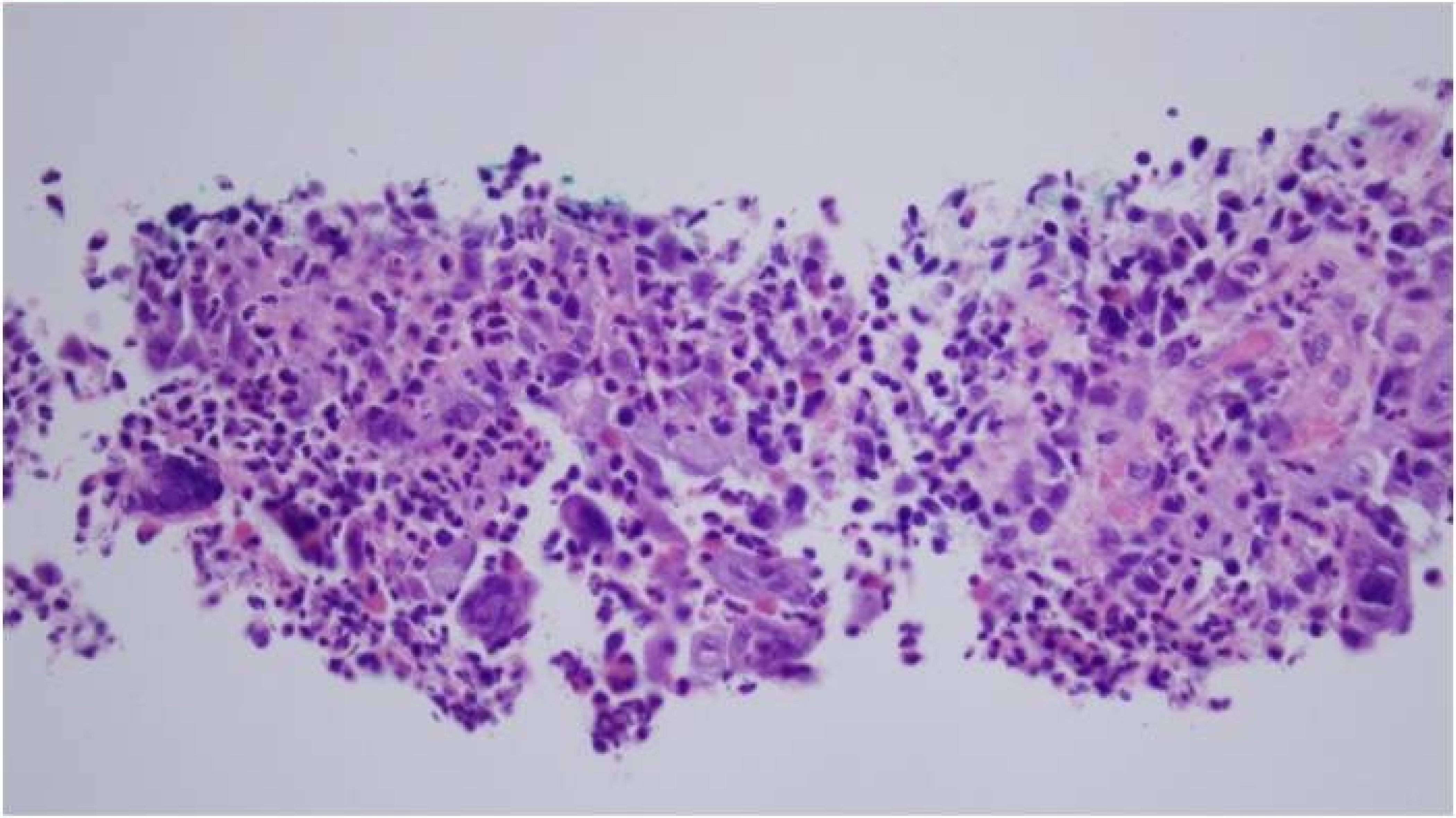
Chickenpox>>

Herpes zoster>> (shingles)

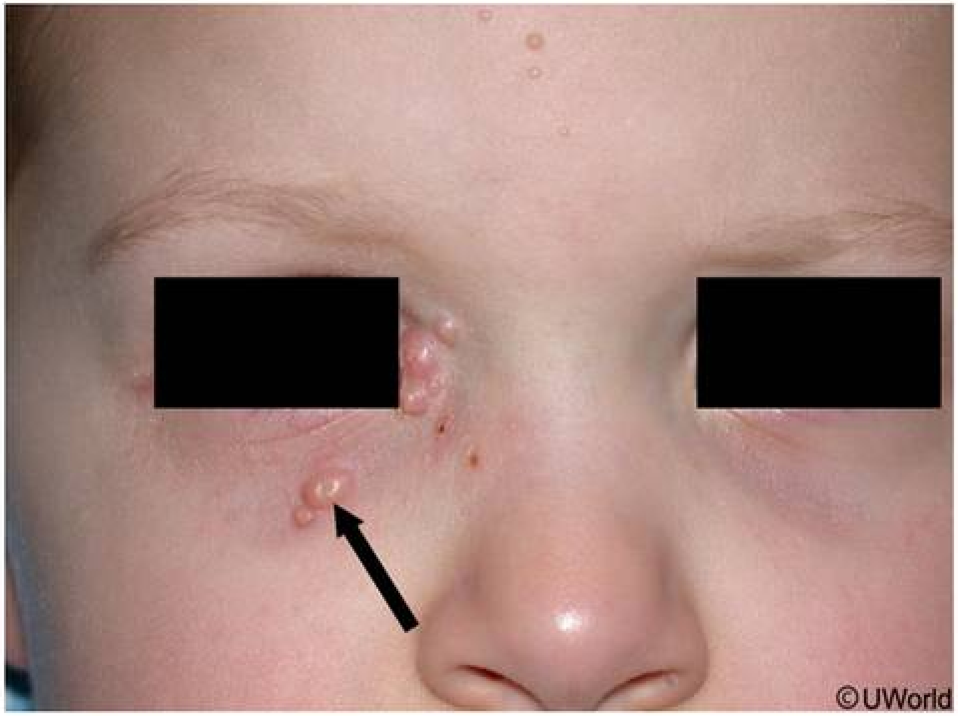
Herpes zoster ophthalmicus>>

VZV infection>> multinucleated giant cells

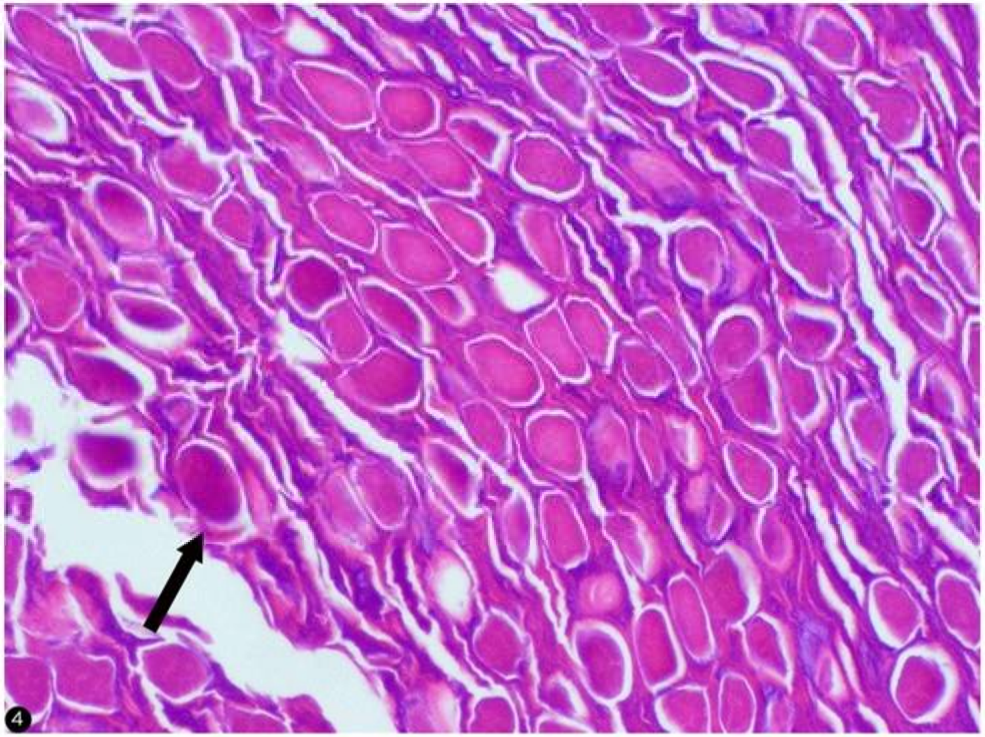


Molluscum contagiosum

Skin colored-umbilicated papules caused by a poxvirus



Molluscum contagiosum>> eosinophilic cytoplasmic inclusions (Molluscum bodies)



HPV strains	Presentation
1-4	Skin warts (verruca vulgaris)
6, 11	Genital warts (condylomata acuminata)
16, 18	Cervical, vaginal, vulvar & anal neoplasia

HPV= human papillomavirus.

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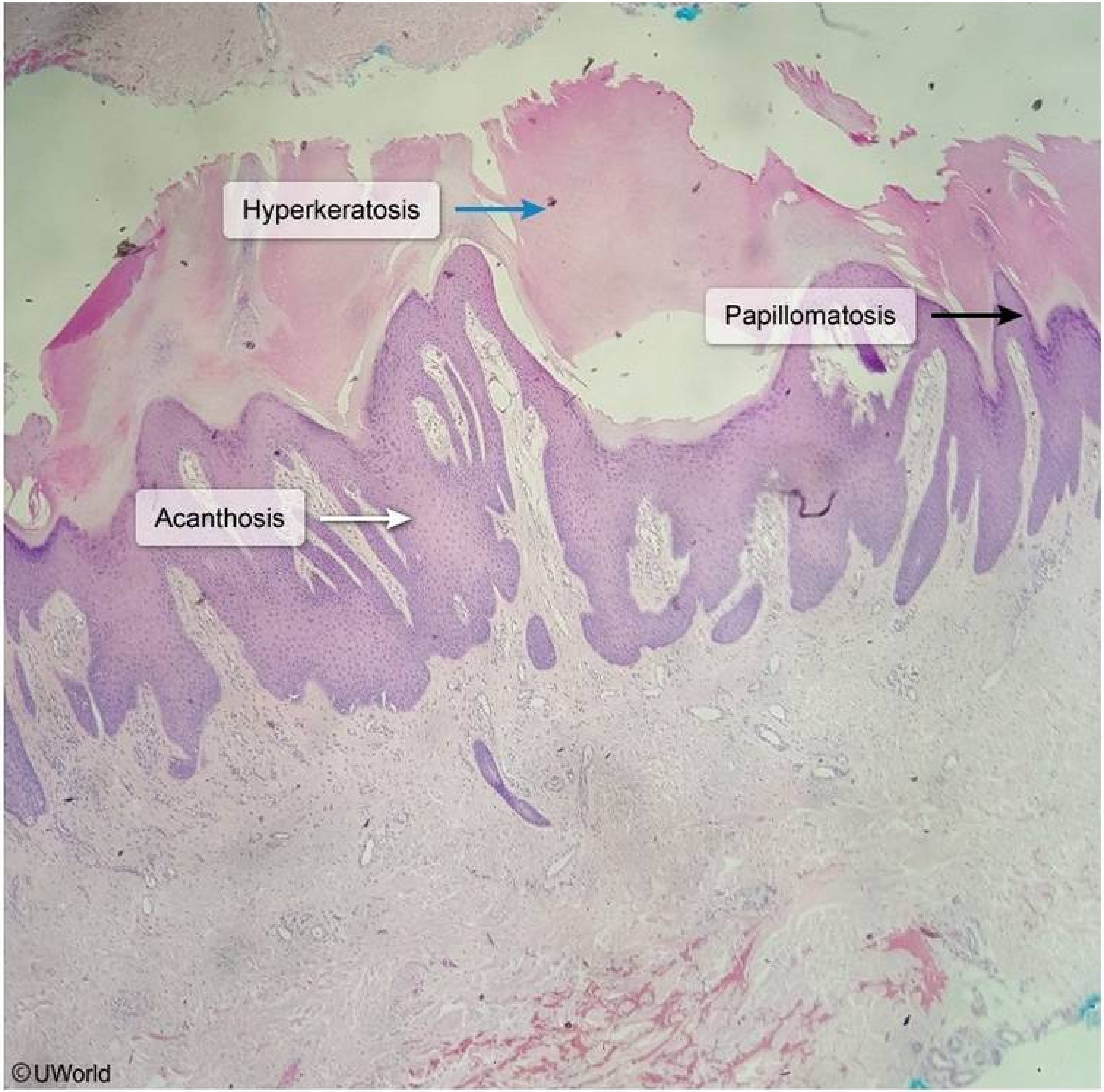
Cutaneous warts (verruca vulgaris)	
Etiology	• Human papillomavirus • Acquired by direct contact
Appearance	• Plaques, papules, verrucous lesions • Skin, nail folds, palms/soles • Thrombosed capillaries on magnified view
Histopathology	• Thickening of stratum corneum • Epidermal hyperplasia & papilloma formation • Cytoplasmic vacuolization (koilocytosis)
Natural history	• Gradual, chronic course • Spontaneous resolution (usually <2 years) • Possible recurrence

Cutaenous warts

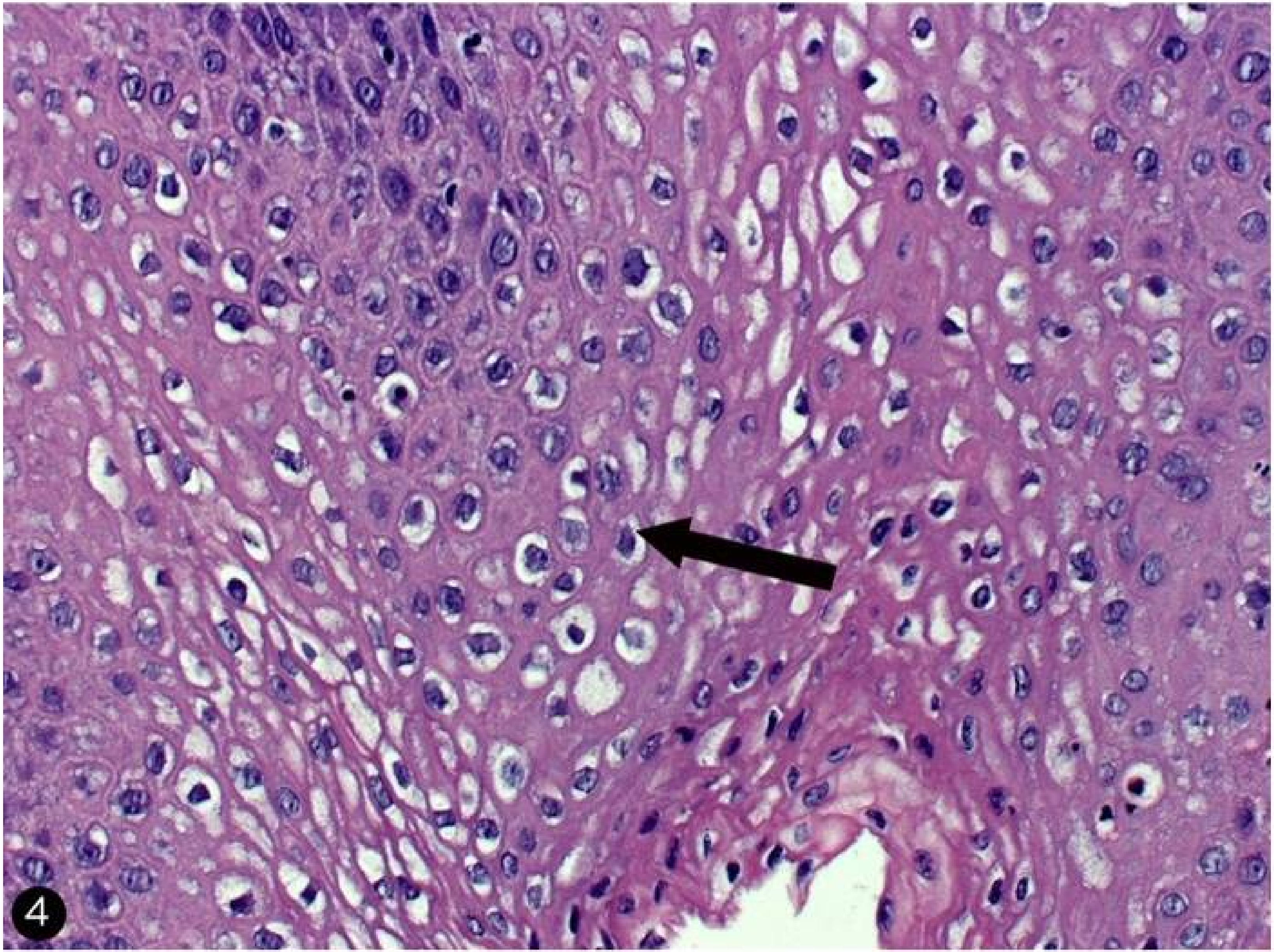
Cutaneous warts

Cutaneous warts>>

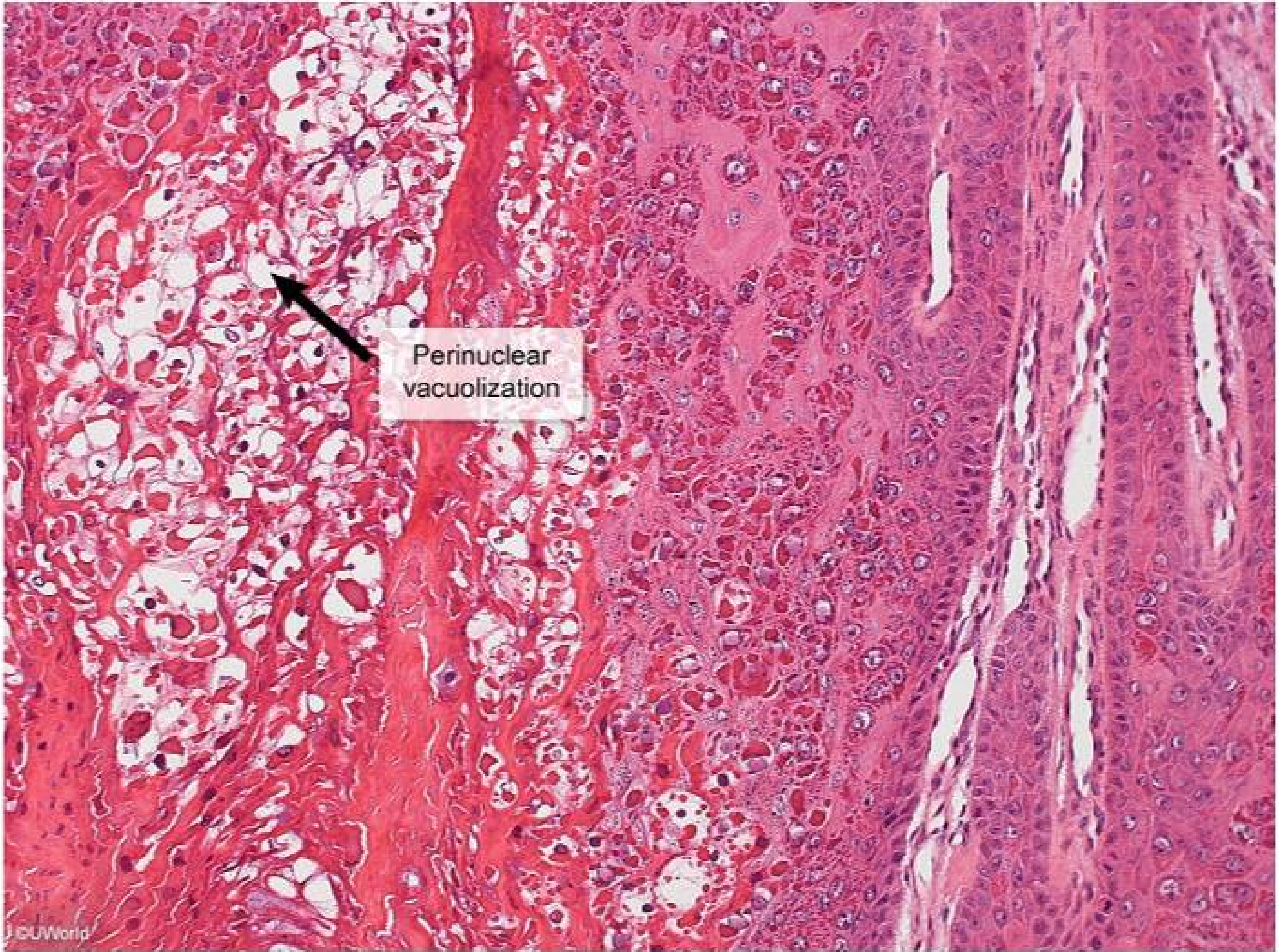


Cutaneous warts>>

koilocytosis>>perinuclear cytoplasmic vacuolization

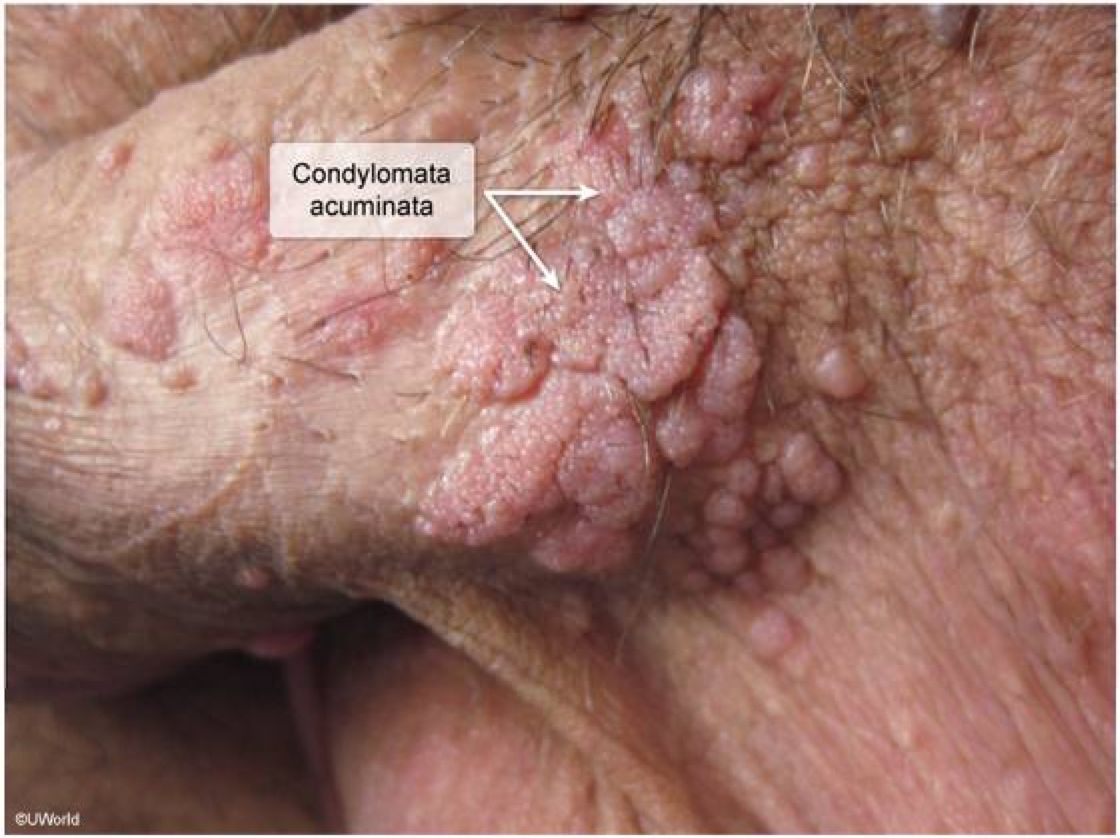


Cutaneous warts

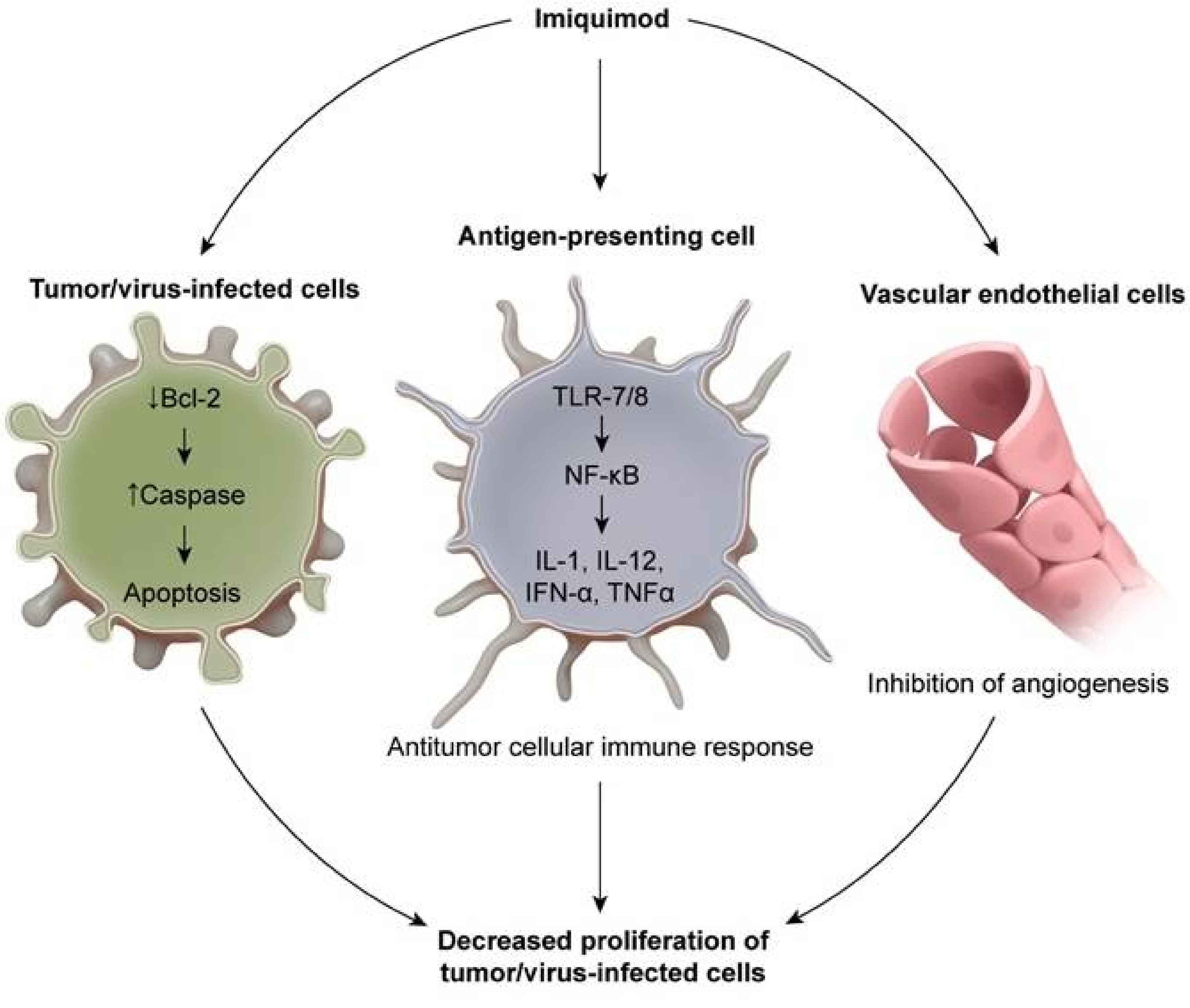


Condylomata acuminata (anogenital warts)	
Etiology	• Human papillomavirus types 6 & 11 • May have coinfection with other genotypes
Appearance	• Plaques, papules, verrucous lesions • Genitalia, perineum, perianal skin
Biopsy	• Thickening of the stratum corneum • Epidermal hyperplasia • Cytoplasmic vacuolization
Disease associations & risk factors	• HIV • Other sexually transmitted infections • Immune suppression
Treatment	• **Chemical:** Podophyllin resin, trichloroacetic acid • **Immunologic:** Imiquimod • **Surgical:** Cryotherapy, laser therapy, excision
Prevention	• Vaccination • Barrier contraception

Condyloma Accuminata (Anogenital Warts)



Imiquimod mechanism of action



Bcl-2 = b-cell lymphoma 2; **IL** = interleukin; **IFN** = interferon; **TNF** = tumor necrosis factor; **TLR** = Toll-like receptor.

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Tinea (dermatophytes)>>

Clinical name for dermatophyte (cutaneous fungal) infections.

Dermatophytes include *Microsporum, Trichophyton,* and *Epidermophyton.*

Branching septate hyphae visible on KOH preparation with blue fungal stain.

Associated with pruritus.

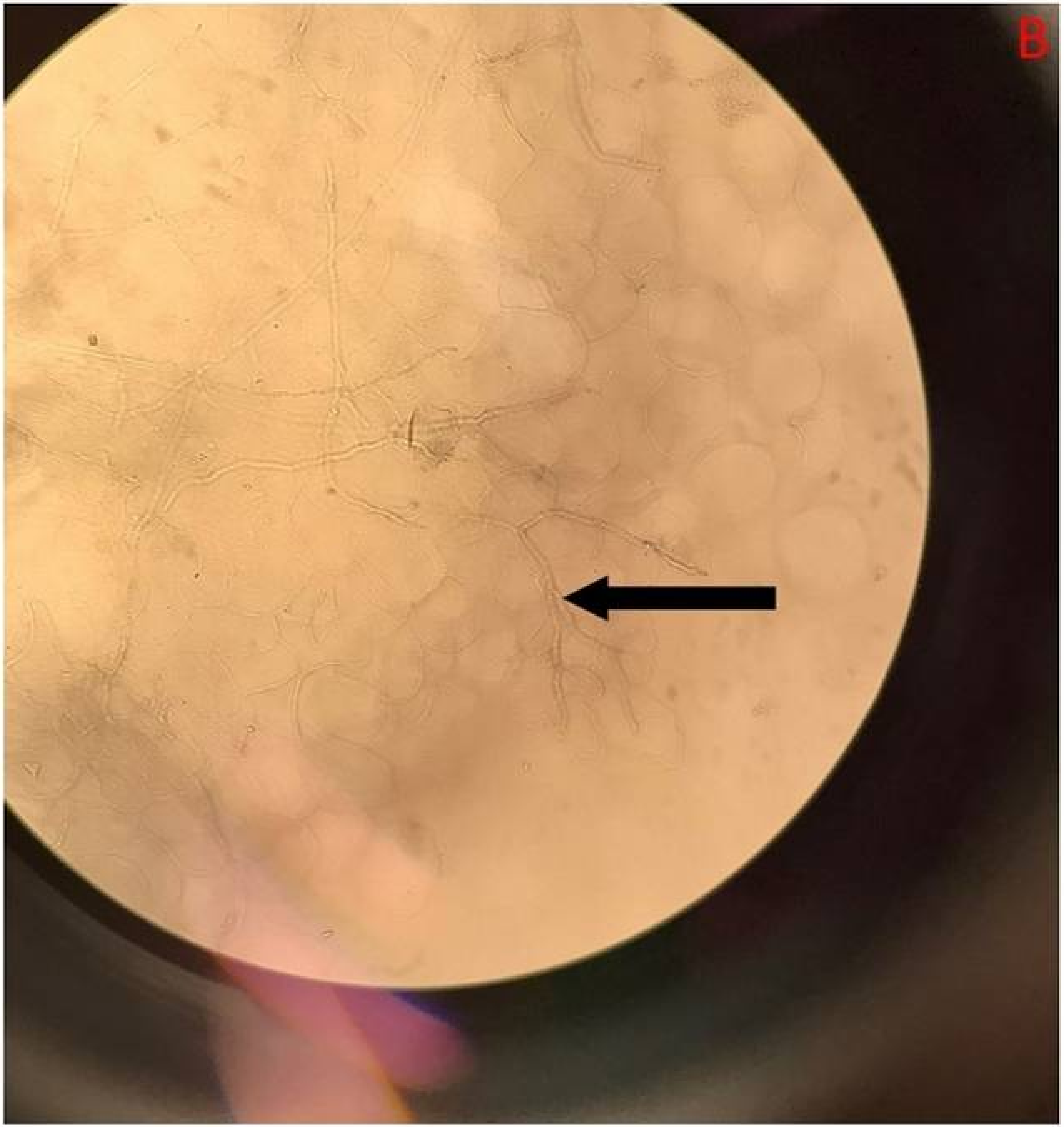
Tinea (dermatophytes)>>

KOH preparation with blue fungal stain

Branching septate hyphae

Dermatophytes>>

microscope of skin scrapings>> branching hyphae



Tinea capitis	
Clinical features	• Scaly erythematous patch on scalp • Hair loss with residual black dot • Possible painful lymphadenopathy • Predominant in African Americans • Human-to-human or fomite (eg, shared combs) transmission
Management	• KOH examination of hair shaft to document spores • Treatment: **Oral griseofulvin (1st line)**, terbinafine, itraconazole, or fluconazole

KOH = potassium hydroxide.

Tinea capitis>>

Tinea corporis (ringworm)	
Risk factors	• Athletes who have skin-to-skin contact • Humid environment • Contact with infected animals (eg, rodents)
Presentation	• Scaly, erythematous, pruritic patch with centrifugal spread • Subsequent central clearing with raised annular border
Treatment	• First-line/localized: Topical antifungals (eg, clotrimazole, terbinafine) • Second-line/extensive: Oral antifungals (eg, terbinafine, griseofulvin)

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Tinea corporis>>

Tinea corporis>>

Tinea cruris>>

Occurs in inguinal area.

Often does not show the central clearing seen in tinea corporis.

Tinea pedis	
Etiology	• *Trichophyton* species (most common)
Clinical features	• Pruritus, erythema, erosions/scaling • Most often interdigital or covering the sole (moccasin type) • Segmented hyphae on KOH preparation
Treatment	• Topical antifungal therapy (eg, azoles, terbinafine) • Keep feet dry & dispose of old footwear

Tinea pedis>>

Interdigital type

Moccasin type

Tinea mannum>> annular appearance

Tinea unguium>>

Onychomycosis; occurs on nails.

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Tinea versicolor (pityriasis versicolor)	
Pathogenesis	*Malassezia globosa* skin flora grows in exposure to hot & humid weather
Clinical features	• Hypopigmented, hyperpigmented, or mildly erythematous lesions (face in children, trunk & upper extremities in adolescents & adults) • +/- Fine scale • +/- Pruritus
Diagnosis	KOH preparation shows hyphae & yeast cells in a "spaghetti & meatballs" pattern
Treatment	Topical ketoconazole, terbinafine, or selenium sulfide

KOH= potassium hydroxide.

Tinea (pityriasis) versicolor>>

Sporotrichosis	
Epidemiology	• *Sporothrix schenckii* (dimorphic **fungus**) • Decaying plant matter/soil • **Gardeners** & landscapers
Clinical	• Subacute/chronic • **Skin papule** → Ulceration with nonpurulent, odorless drainage • **Proximal lesions** along lymphatic chain • LAD, deeper spread & systemic symptoms are **rare**
Diagnostic	• Cultures (aspirate fluid or biopsy) >> **cigar**-shaped yeast
Treatment	• 3-6 months of oral **itraconazole**

Sporotrichosis>> ascending lymphangitis

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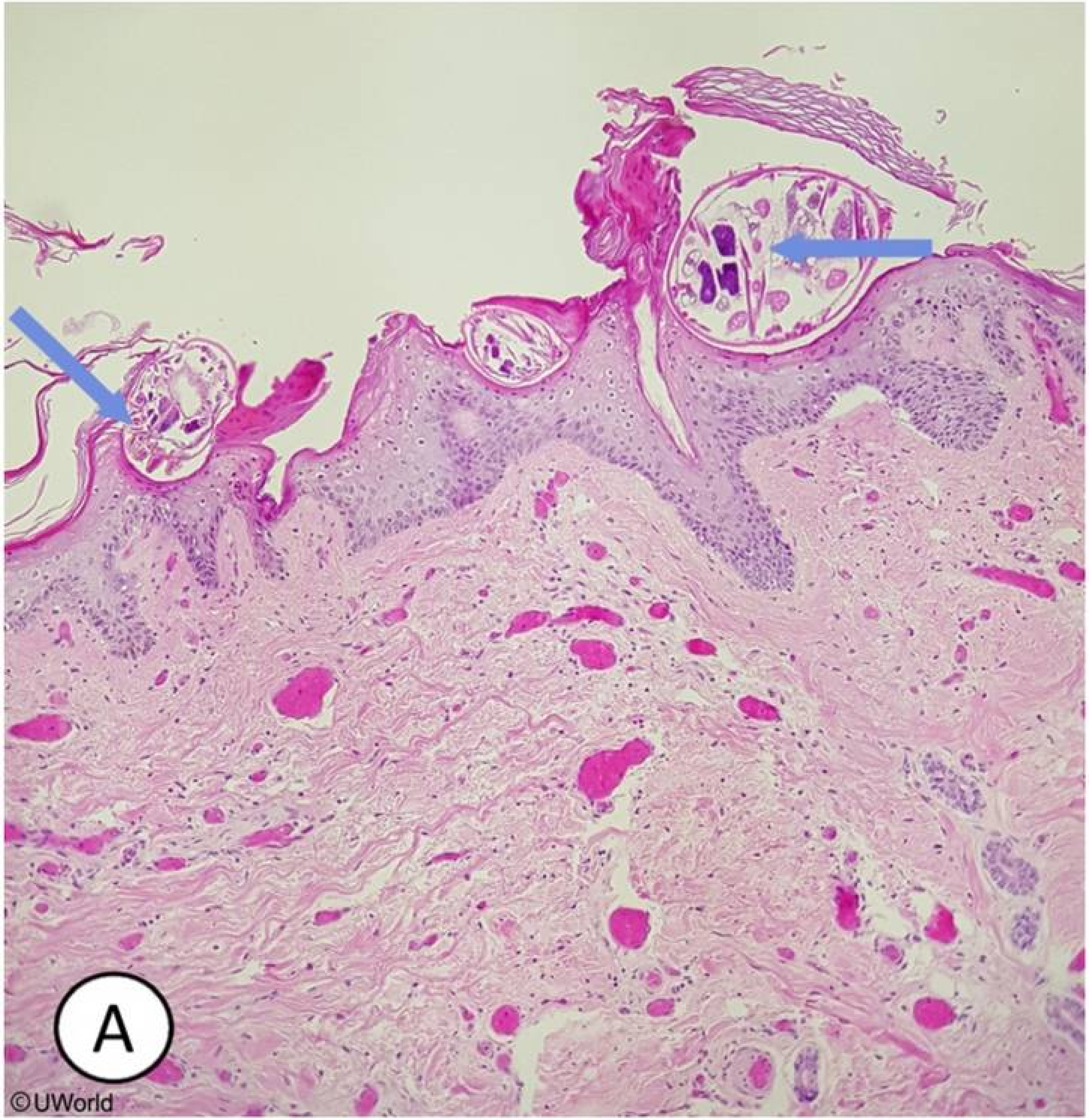
Sarcoptes scabiei>>

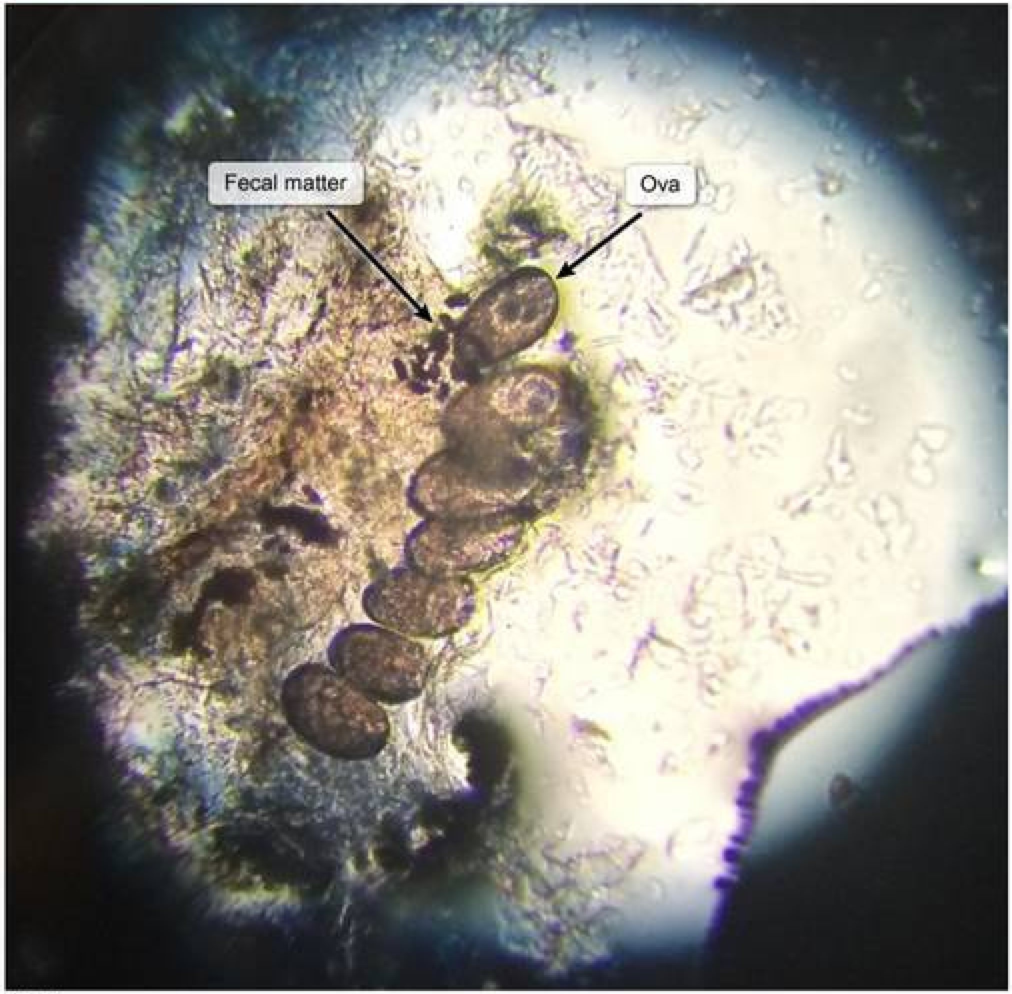
Mites burrow into stratum corneum and cause **scabies**—pruritus (worse at night) and serpiginous burrows (lines) often between fingers and toes.	Common in children, crowded populations (jails, nursing homes); transmission through skin-to-skin contact (most common) or via fomites. Treatment: permethrin cream, washing/drying all clothing/bedding, treat close contacts.

Scabies—serpiginous burrows (lines) often between fingers and toes.

Scabies>> visible mite's internal organs



Scabies>> **mite.. ova.. feces**



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Pediculus humanus/ Phthirus pubis>>

Blood-sucking lice that cause intense pruritus with associated excoriations, commonly on scalp and neck (head lice), waistband and axilla (body lice), or pubic and perianal regions (pubic lice).	Body lice can transmit *Rickettsia prowazekii* (epidemic typhus), *Borrelia recurrentis* (relapsing fever), *Bartonella quintana* (trench fever). Treatment: pyrethroids, malathion, or ivermectin lotion, and nit combing. Children with head lice can be treated at home without interrupting school attendance.

***Phthirus pubis*>>** pubic lice

Phthirus pubis>> with crab-like claws

Autoimmune blistering skin disorders		
	Pemphigus vulgaris	**Bullous pemphigoid**
Autoantibody target	• Desmosomes (desmoglein 3 & 1)	• Hemidesmosomes (bullous pemphigoid antigens 1 & 2)
Histopathology	• **Intraepidermal** cleavage • **Acantholysis** (detached keratinocytes) • "Tombstone cells" along basal layer	• **Subepidermal** cleavage • No acantholysis
Immunofluorescence	• Netlike intercellular IgG	• Linear IgG at basement membrane
Clinical features	• Middle-aged or elderly • **Flaccid** bullae • Oral/mucosal involvement • Nikolsky sign positive	• Predominantly elderly • **Tense** bullae • Rare oral/mucosal involvement

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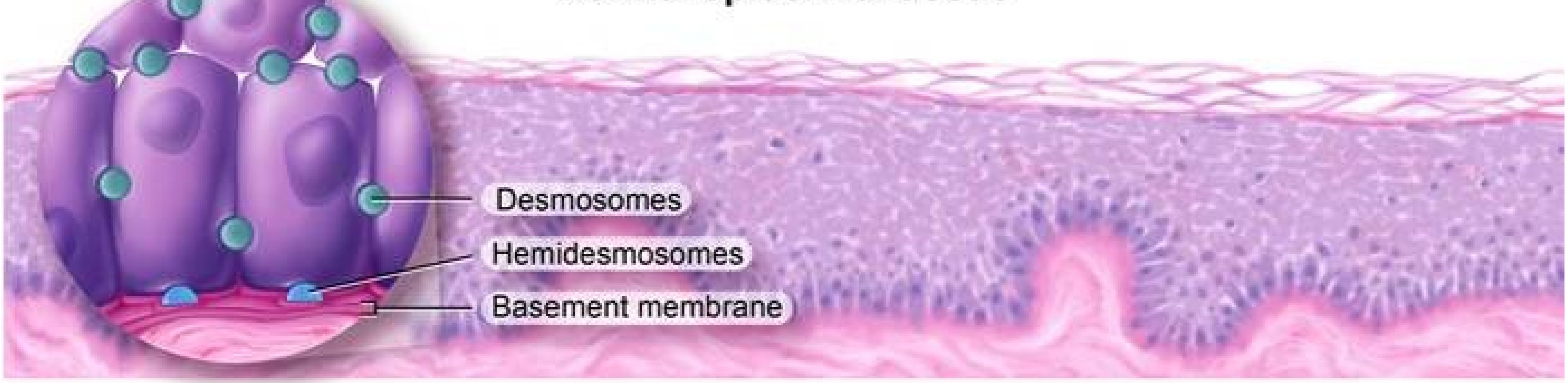
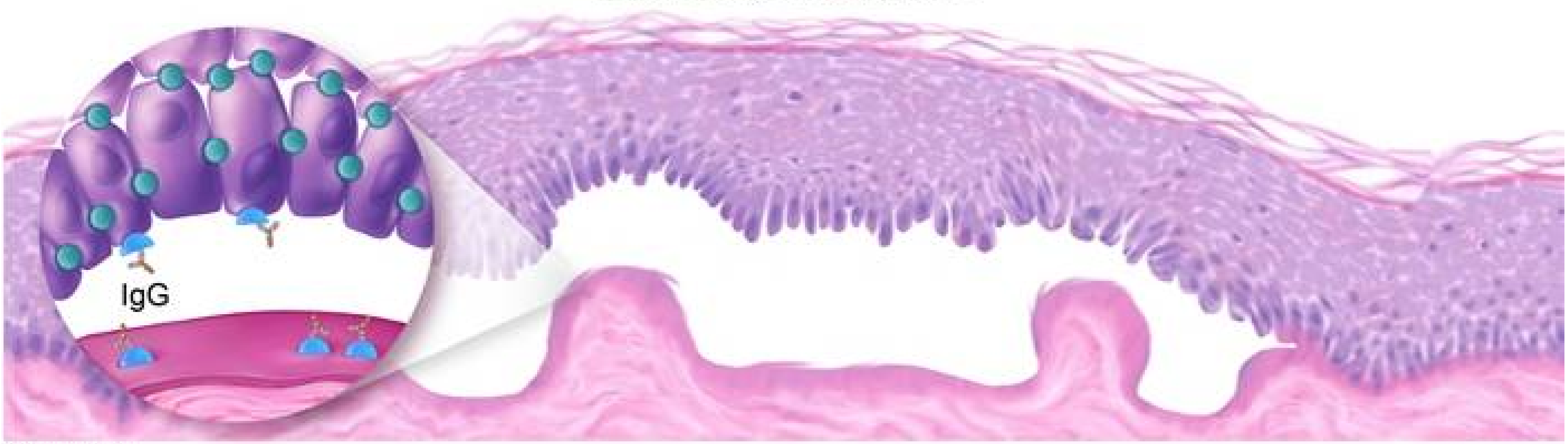
Normal epidermal tissue



Pemphigus vulgaris

Bullous pemphigoid



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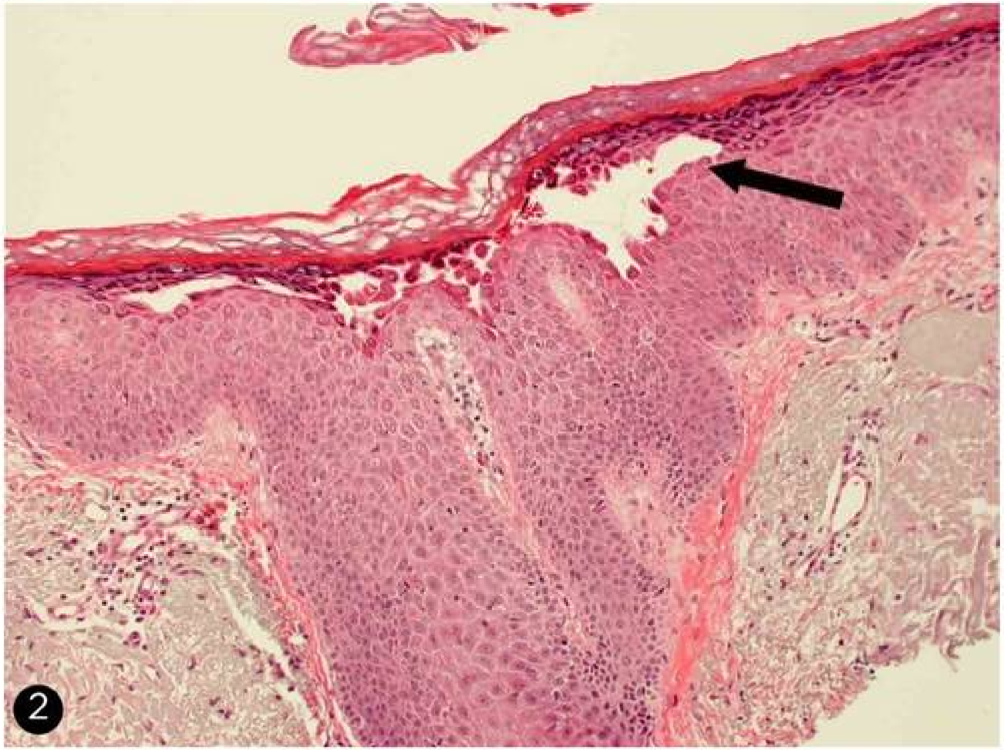
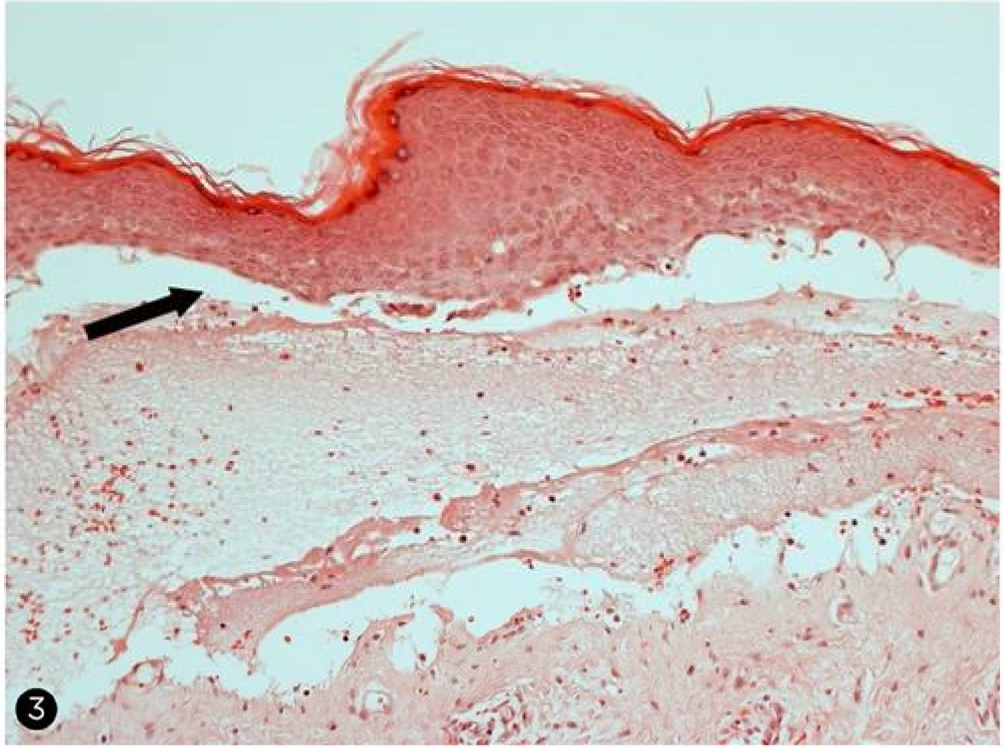
Pemphigus vulgaris>> intraepidermal cleavege



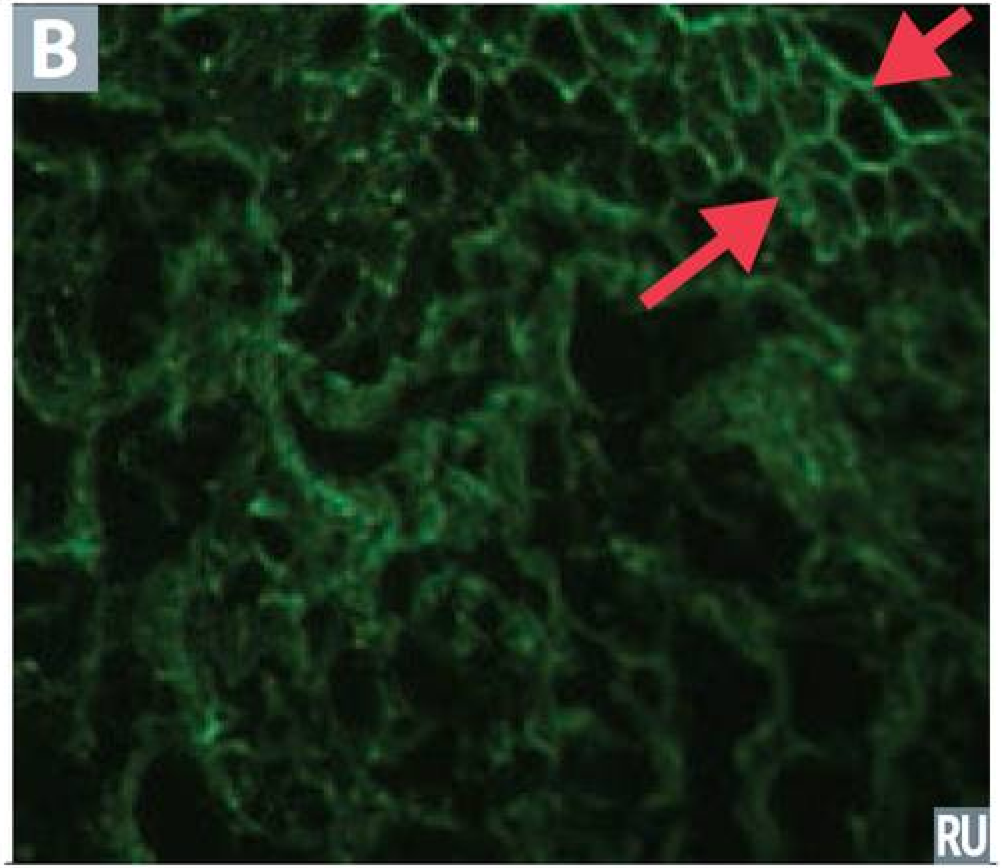
Bullous pemphigoid>> subepidermal cleavege



Immunofluorescence

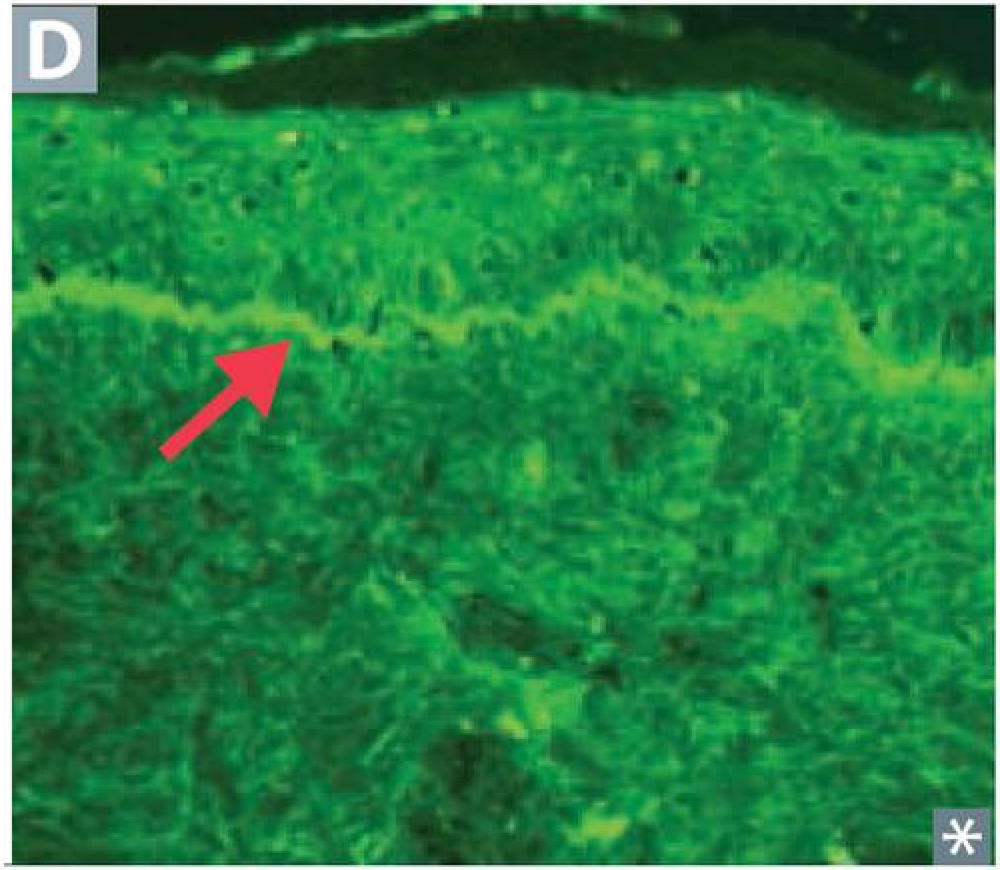
Pemphigus vulgaris>>

Intarepidermal reticular pattern



Bullous pemphigoid>>

Linear pattern at epidermal-dermal junction



Pemphigus vulgaris>> Flaccid bullae

Bullous pemphigoid>> Tense bullae

Epidermolysis bullosa	
Etiology	• Inherited disorders • Mutations involving intraepidermal & dermoepidermal adhesion complexes
Clinical features	• Onset in infancy/childhood • Bullae, erosions & ulcers • Lesions triggered by friction or minor trauma • Histopathology: intraepidermal cleavage most common
Complications	• Malnutrition • Infection

Epidermolysis bullosa>> ulcer

Dermatitis herpetiformis>>

Pruritic papules, vesicles, and bullae (often found on elbows, knees, buttocks). Deposits of IgA at tips of dermal papillae. Associated with celiac disease.

Treatment: dapsone, gluten-free diet.

Dermatitis herpetiformis

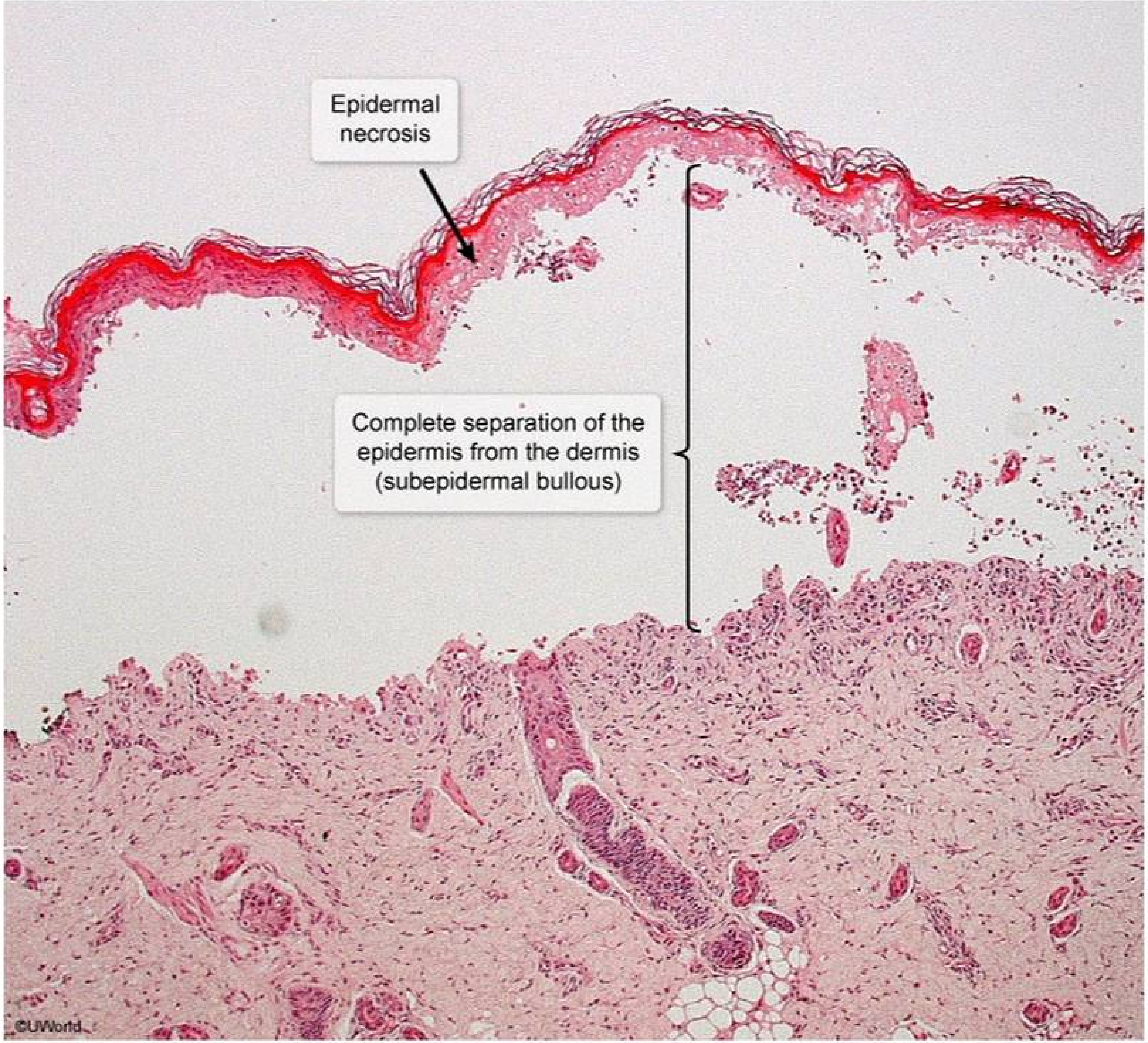
Erythema multiforme>>

Associated with infections (eg, *Mycoplasma pneumoniae*, HSV), drugs (eg, sulfa drugs, β-lactams, phenytoin). Presents with multiple target lesions

Stevens-Johnson syndrome & toxic epidermal necrolysis	
Nomenclature	<10% of BSA: SJS 10%-30%: SJS/TEN overlap >30%: TEN
Clinical features	• 4-28 days after exposure to trigger (2 days after repeat exposure) • Acute influenza-like **prodrome** • Rapid-onset erythematous macules, vesicles, bullae • Necrosis & **sloughing of skin at dermal-epidermal junction (⊕ Nikolsky)** • **Mucosal involvement**
Common triggers	**Drugs** • Allopurinol • Antibiotics (eg, sulfonamides) • Anticonvulsants (eg, carbamazepine, lamotrigine, phenytoin) • NSAIDs (eg, piroxicam) • Sulfasalazine **Other** • *Mycoplasma pneumoniae* • Vaccination • Graft-vs-host disease

BSA = body surface area; **NSAIDs** = nonsteroidal anti-inflammatory drugs; **SJS** = Stevens-Johnson syndrome; **TEN** = toxic epidermal necrolysis.

SJS/TEN>>



Epidermal necrosis
Complete separation of the epidermis from the dermis (subepidermal bullous)
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SJS/TEN>>

Acanthosis nigricans>>

Epidermal hyperplasia causing symmetric, hyperpigmented thickening of skin, especially in axilla or on neck.

Associated with **insulin resistance** (eg, diabetes, obesity, Cushing syndrome, PCOS), **visceral malignancy** (eg, gastric adenocarcinoma).

Acrochordon (Skin tag)>>

Actinic keratosis>>

Premalignant lesions caused by sun exposure.
Small, **rough**, erythematous or brownish papules or plaques.
Risk of **squamous cell carcinoma** is proportional to degree of epithelial dysplasia.

Actinic keratoses>>

Cutaneous horn

Erythema nodosum>>

Painful, raised inflammatory lesions of subcutaneous fat (panniculitis), usually on anterior shins.
Often idiopathic, but can be associated with sarcoidosis, coccidioidomycosis, histoplasmosis, TB, streptococcal infections, leprosy, inflammatory bowel disease.

Lichen planus	

Clinical findings	• **5 "Ps":** pruritic, purple/pink, polygonal, papules & plaques
	• Lacy, white network of lines (Wickham striae)
	• **Locations:**
	  ○ Skin (eg, ankles, wrists)
	  ○ Oral mucosa (white papules & plaques ± erythema, mucosal atrophy, ulcers)
	  ○ Genitalia

Pathologic findings	• Hyperkeratosis • Lymphocytic interface dermatitis • Eosinophilic colloid (Civatte) bodies • Thickened stratum granulosum, sawtooth rete ridges
Natural history	• Chronic symptoms • Formation of lesions at sites of trauma (Köbner phenomenon) • Resolution within 2 yr (mucosal lesions may persist/recur)

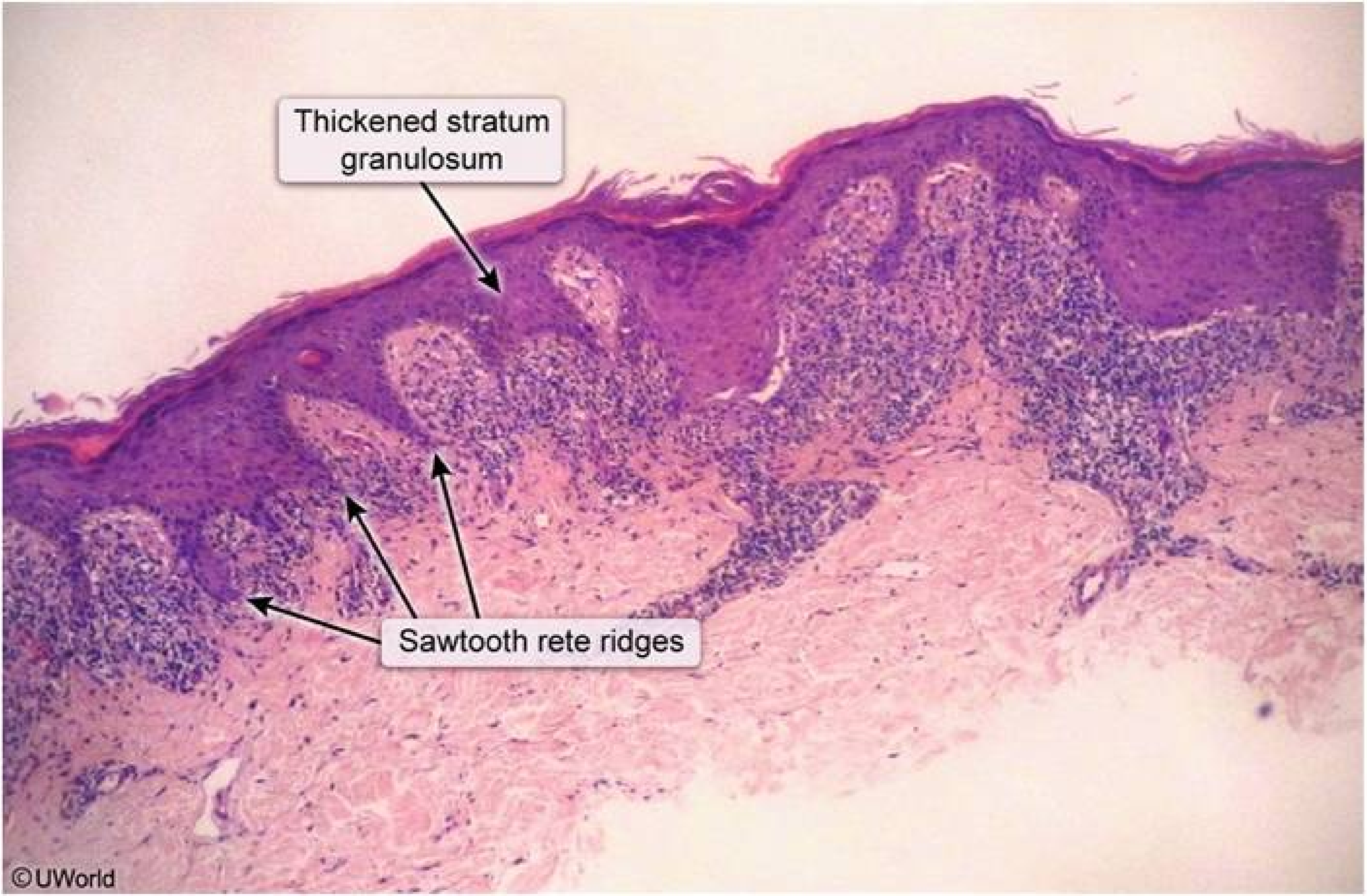
5 P's of lichen Planus

Lichen Planus>>

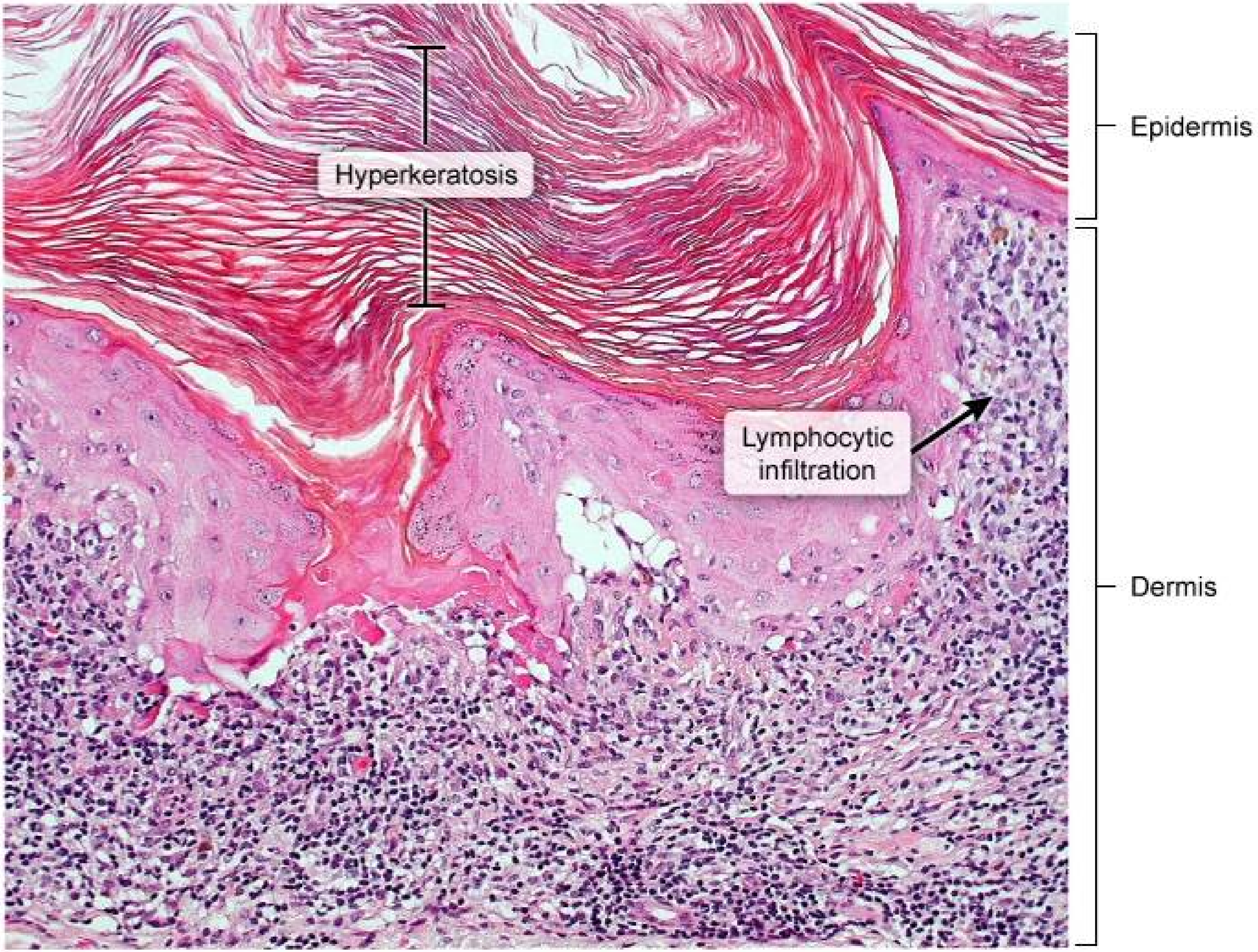
Wickham striae (reticular white lines)

Lichen Planus>>

Hypergranulosis and sawtooth infiltrate of lymphocytes at dermal-epidermal junction



Lichen planus



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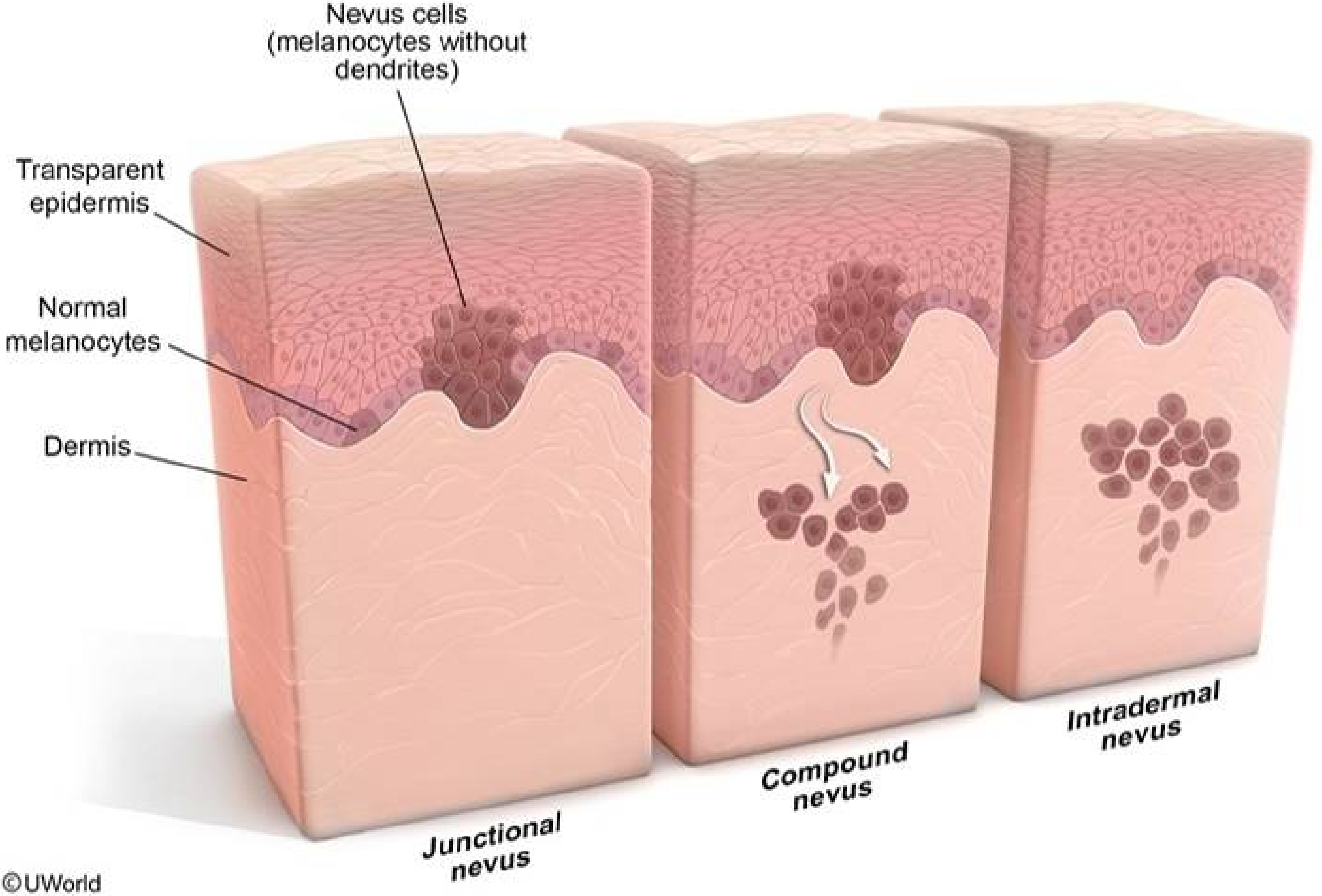
Melanocytic nevus>>

Common mole. Benign, but melanoma can arise in congenital or atypical moles. **Intradermal nevi** are papular. **Junctional nevi** are flat macules.

Histology of nevi



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Junctional nevi>>

Intradermal nevi>>

Congenital nevi>>

Atypical nevi>>

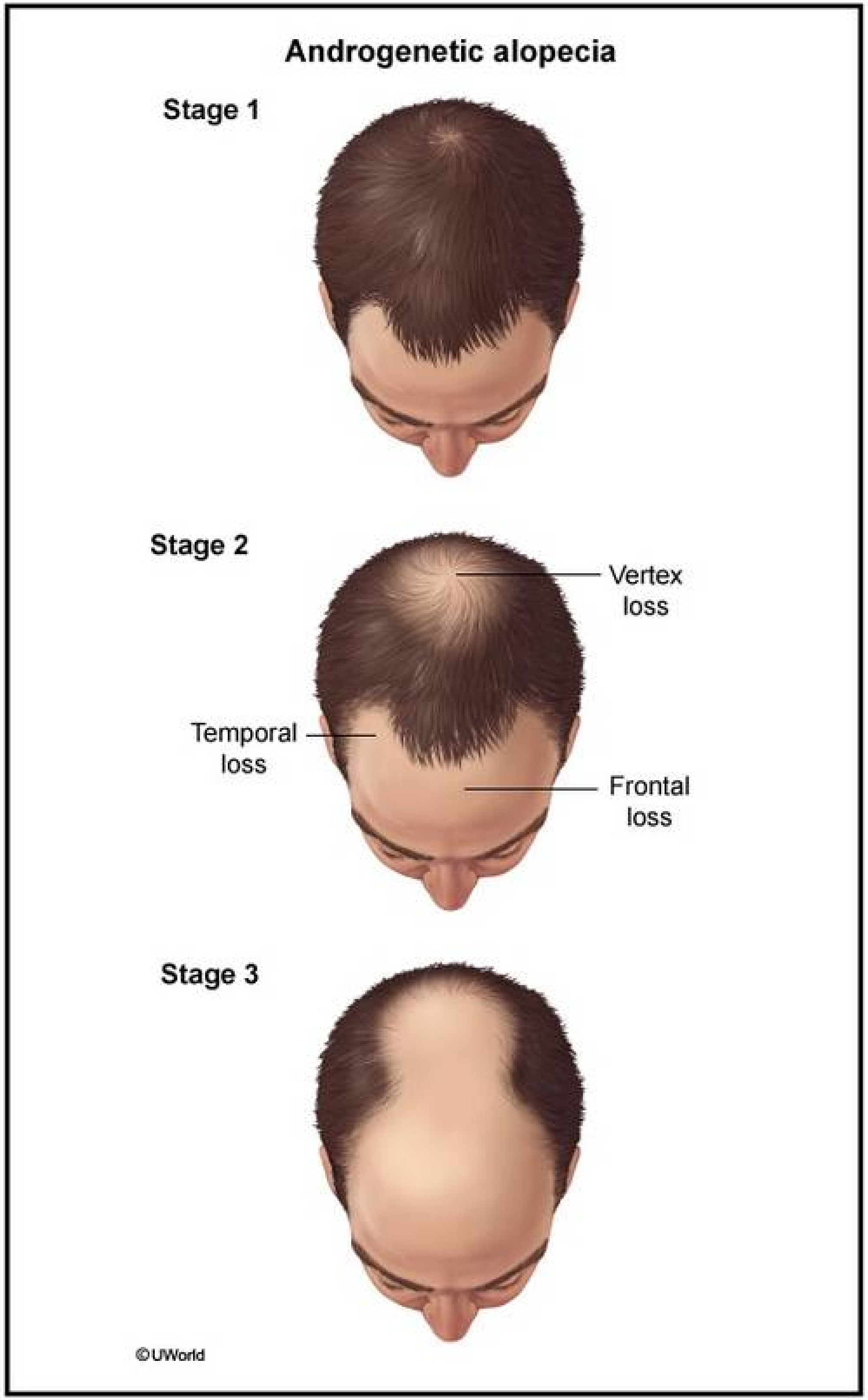
Pseudofolliculitis barbae

Foreign body inflammatory facial skin disorder characterized by firm, hyperpigmented papules and pustules that are painful and pruritic. Located on cheeks, jawline, and neck. Commonly occurs as a result of shaving ("razor bumps"), primarily affects African-American males.

Pityriasis rosea>>

"**Herald patch**" (**A**) followed days later by other scaly erythematous plaques, often in a "**Christmas tree**" distribution on trunk (**B**) . Multiple pink plaques with collarette scale. Self-resolving in 6–8 weeks.

Androgenetic (male pattern) alopecia in men	
Pathophysiology	• Genetic factors + androgen (dihydrotestosterone) effect • Shortened anagen phase • Follicular miniaturization
Clinical features	• Onset at puberty, progressive course • Hair loss at temporal, frontal, vertex scalp • Shorter, thinner, sparser hair
Treatment	• Minoxidil • 5-alpha reductase inhibitors (eg, finasteride)


Androgenetic alopecia
Stage 1
Stage 2
Vertex loss
Temporal loss
Frontal loss
Stage 3
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Sunburn>>

Acute cutaneous inflammatory reaction due to excessive UV irradiation.
Causes DNA mutations, inducing apoptosis of keratinocytes.
UV**B** is dominant in sun**B**urn, UV**A** in t**A**nning and photo**A**ging.
Exposure to UVA and UVB increase risk of skin cancer.

Sunburn	

Prevention	• Remain indoors between 10 AM-4 PM
	• Wear protective clothing:
	  ◦ Hats, pants, long-sleeved shirts
	  ◦ Tightly woven, thick, or dark-colored fabrics
	• Apply sunscreen 30 minutes before sun exposure
	• Avoid tanning beds

Clinical manifestation	• Symptoms begin hours after exposure, resolve by day 3 - 7 • **Mild-moderate**: Erythema, tenderness • **Sever:** As above **+** blistering, systemic symptoms (e.g, fever, vomiting, headache)

Treatment	• Mild-moderate sunburn:
	  ◦ Topical: Cool compresses, calamine lotion, aloe vera
	  ◦ Oral: NSAIDs
	• Severe sunburn: Hospitalization
	  ◦ Intravenous fluids & analgesia
	  ◦ Wound care

Complications	• Cancer:
	  ◦ Melanoma
	  ◦ Basal cell carcinoma
	  ◦ Squamous cell carcinoma
	• Photoaging

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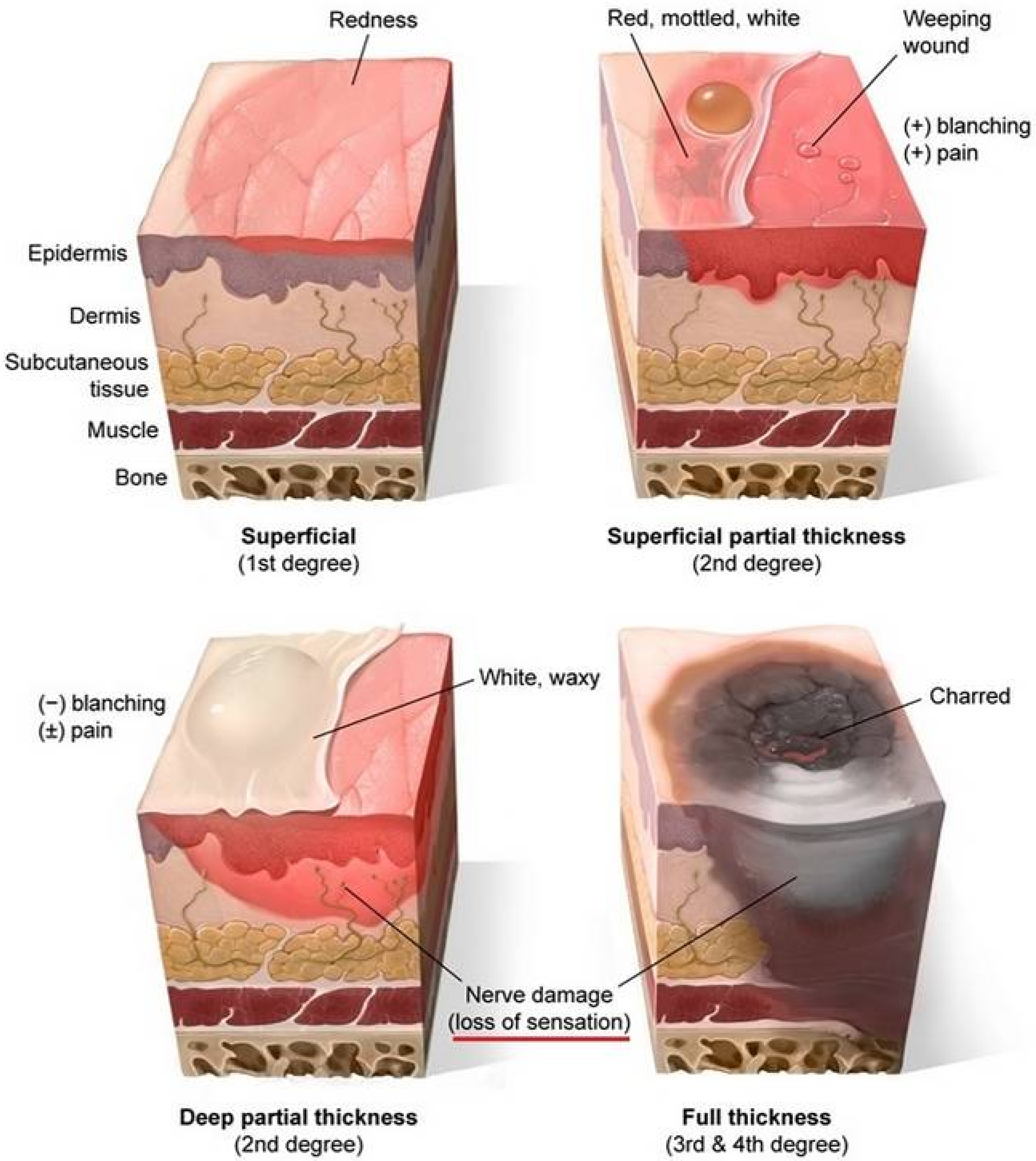
Burn classification



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The earliest morphologic change that occurs after a superficial thermal burn is erythema due to the release of preformed mediators (eg, histamine) from mast cells. Deeper (eg, partial-thickness) burn wounds form blisters due to fluid extravasation through gaps between damaged venule endothelial cells.

Skin cancer>>

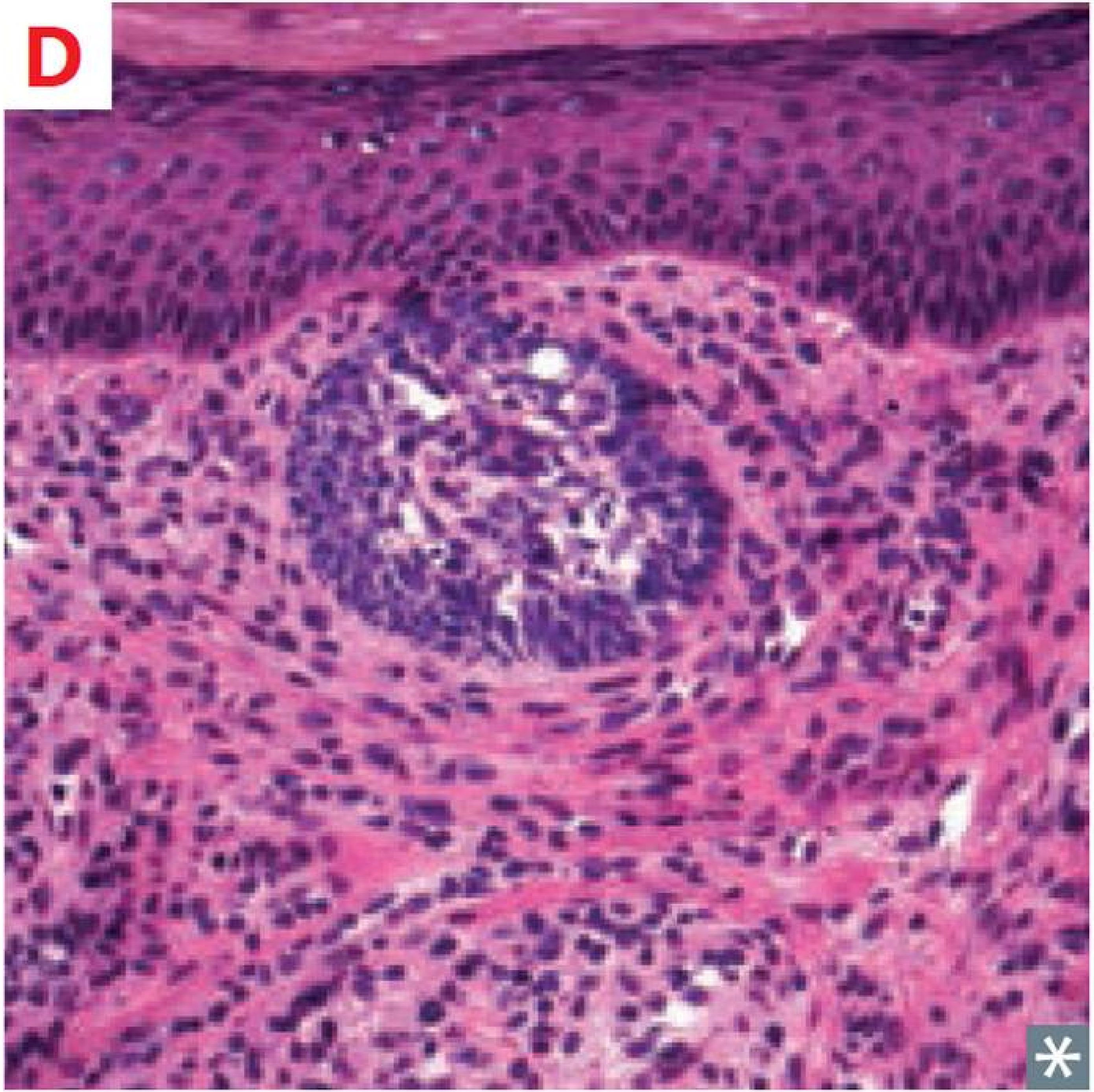
Basal cell carcinoma more common above **upper lip**
Squamous cell carcinoma more common below **lower lip**
Sun exposure strongly predisposes to skin cancer.

Basal cell carcinoma>>

Most common skin cancer. Found in sun-exposed areas of body (eg, face). Locally invasive, but rarely metastasizes. Waxy, pink, pearly nodules, commonly with telangiectasias, **rolled borders** **A** , central crusting or ulceration. BCCs also appear as **nonhealing ulcers** with infiltrating growth **B** or as a **scaling plaque** (superficial BCC) **C**

Basal cell tumors>>

"palisading" (aligned) nuclei D



Keratoacanthoma>>

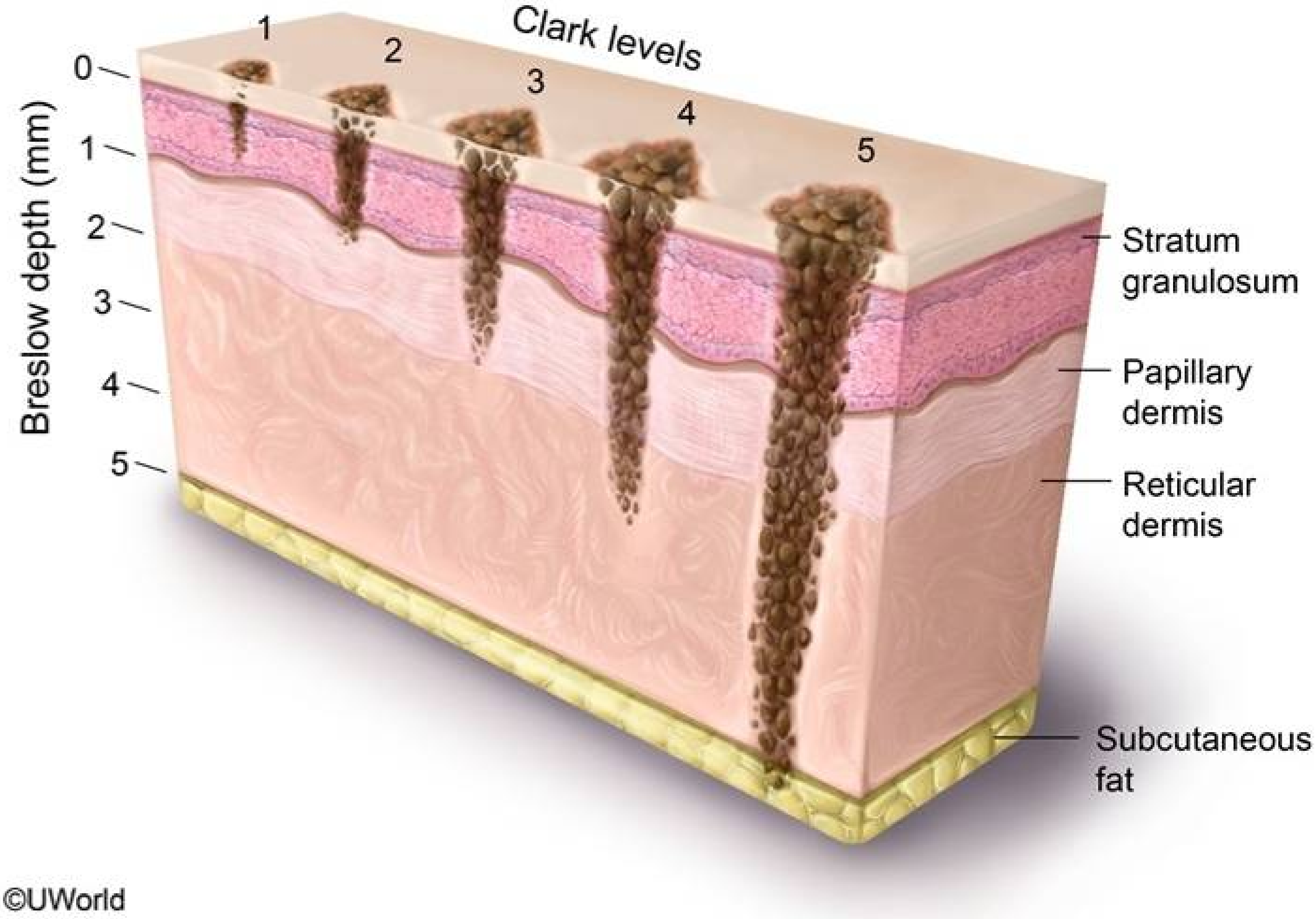
Seen in middle-aged and elderly individuals.
Rapidly growing, resembles **squamous cell carcinoma**.
Presents as dome-shaped nodule with keratin-filled center.
Grows rapidly (4-6 weeks) and may spontaneously regress.

Melanoma>>

Common tumor with significant risk of metastasis. **S-100** tumor marker. Associated with dysplastic nevi; fair-skinned persons are at increased risk.

Melanoma depth of invasion

Depth of tumor>> correlates with risk of metastasis



Clinical features of melanoma (ABCDEs)
• **A**symmetry: When bisected, the 2 sides are not identical • **B**order irregularities: Uneven edges, pigment fading off • **C**olor variegation: Variable mixtures of brown, tan, black & red • **D**iameter: ≥6 mm • **E**volving: Lesion changing in size, shape, or color; new lesion

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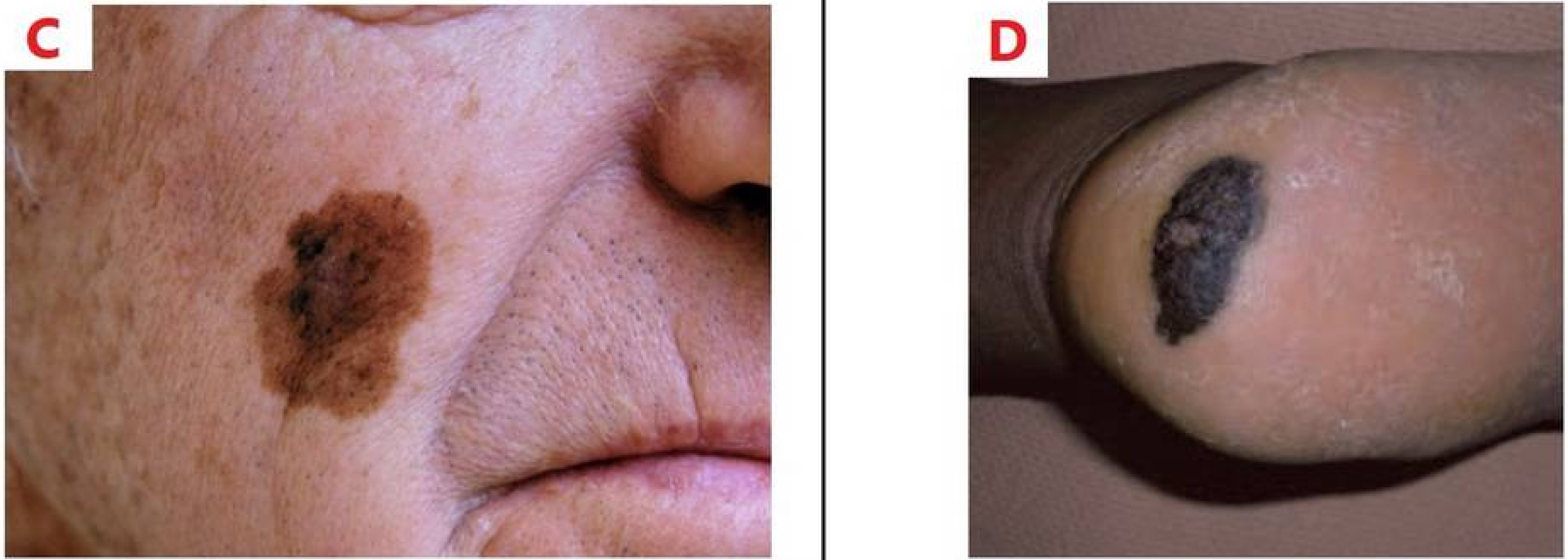
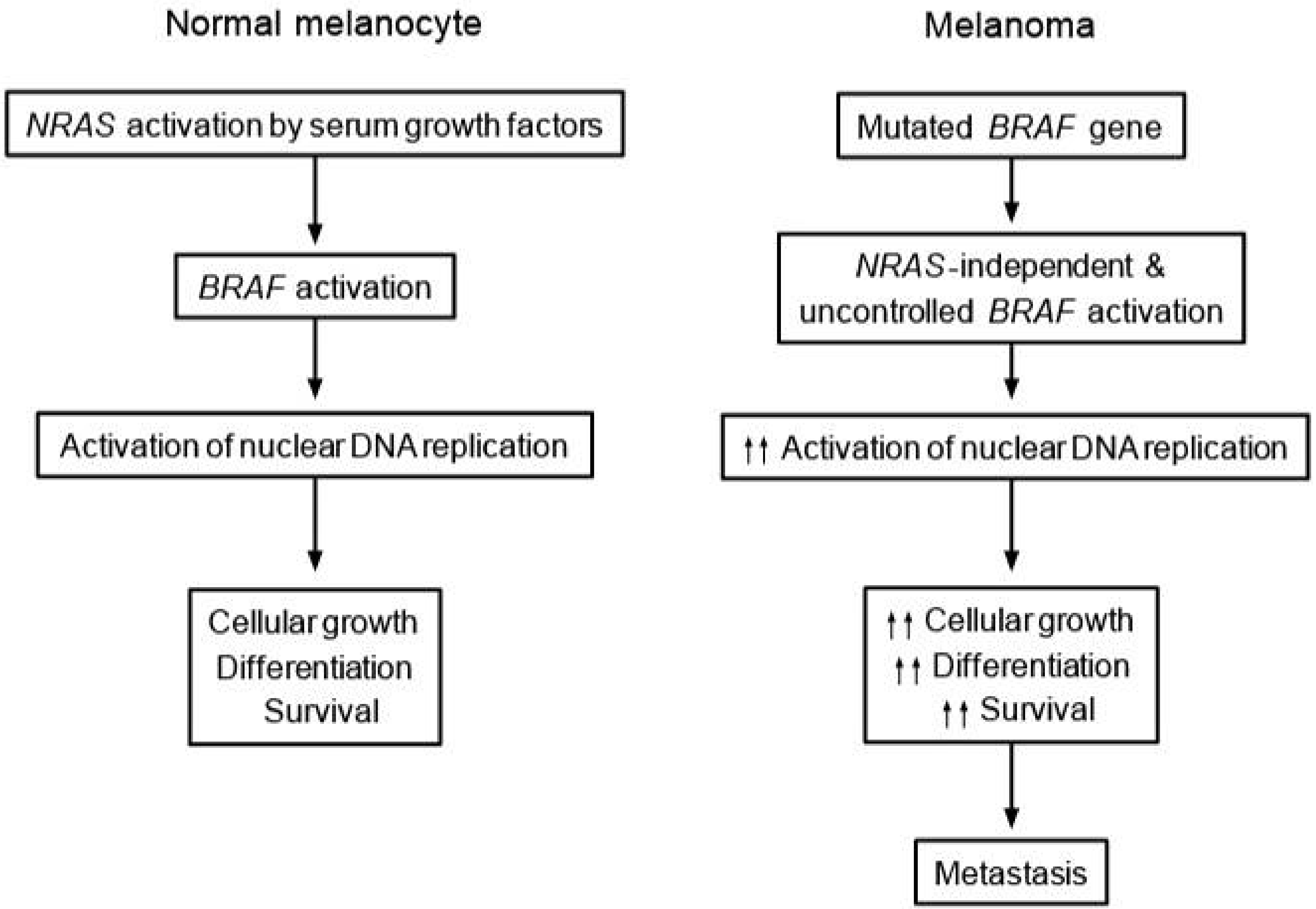
4 different types of melanoma:

superficial spreading **A** , nodular **B**, lentigo maligna **C**, and acral lentiginous (highest prevalence in African-Americans and Asians) **D**


C
D



Normal melanocyte
NRAS activation by serum growth factors
BRAF activation
Activation of nuclear DNA replication
Cellular growth
Differentiation
Survival
Melanoma
Mutated *BRAF* gene
NRAS-independent & uncontrolled *BRAF* activation
↑↑ Activation of nuclear DNA replication
↑↑ Cellular growth
↑↑ Differentiation
↑↑ Survival
Metastasis

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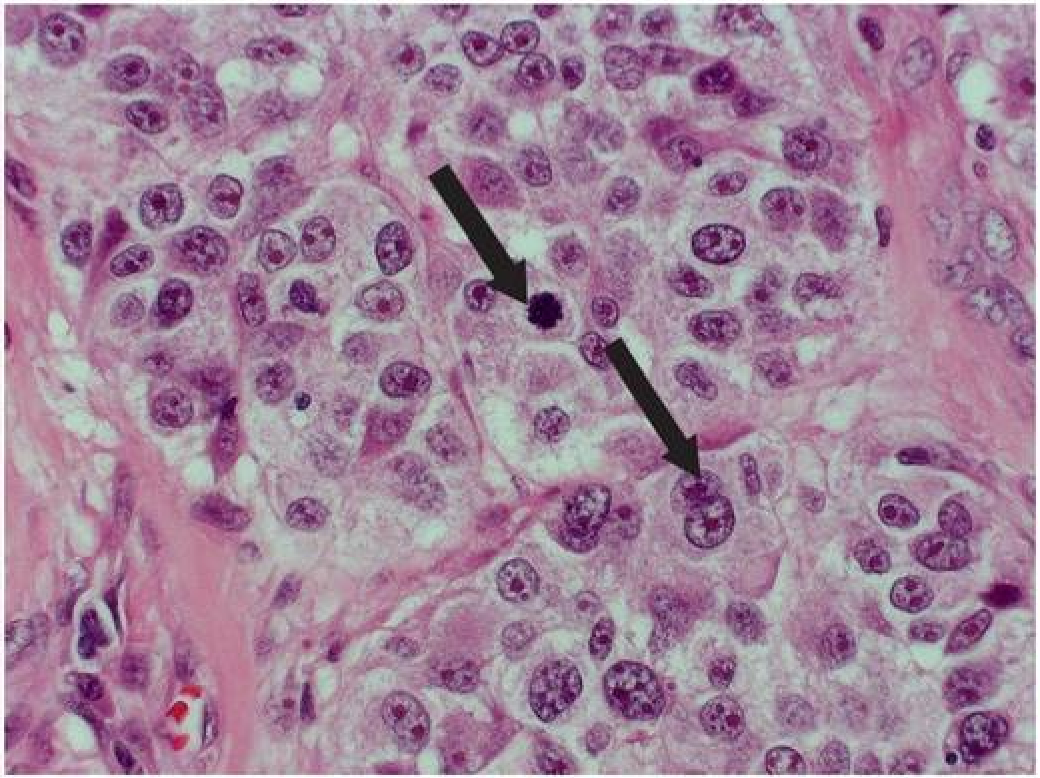
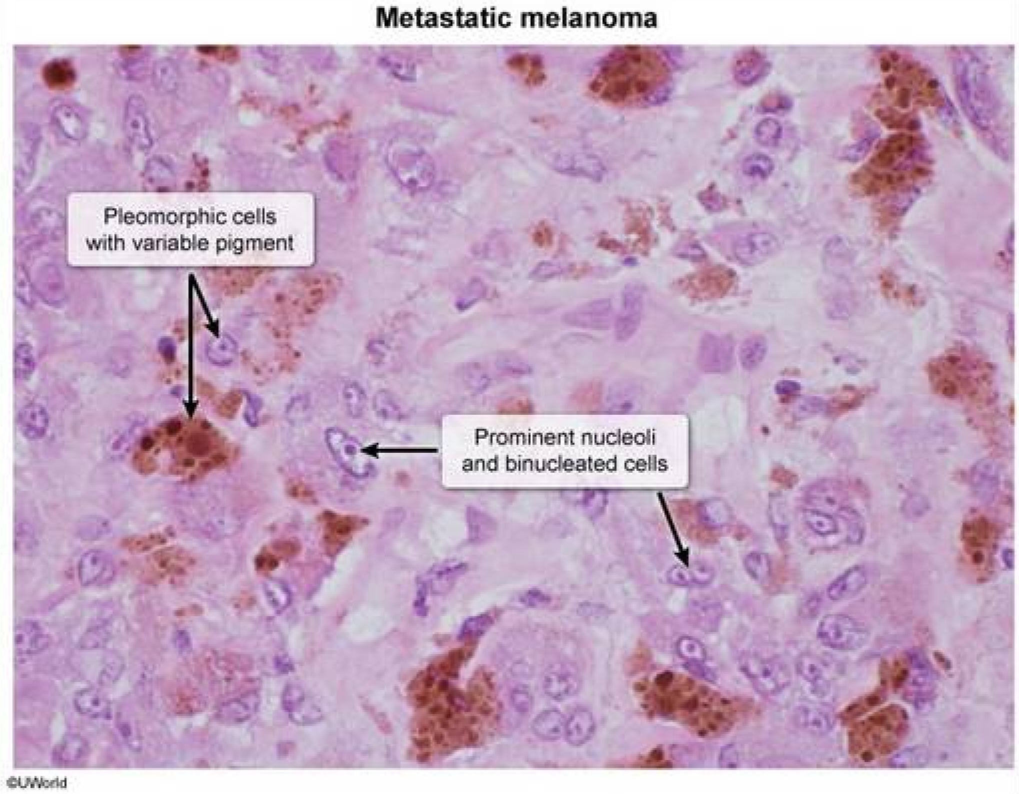
Melanoma>>

Pleomorphic cells with variable pigment, prominent nucleoli and binucleated cells

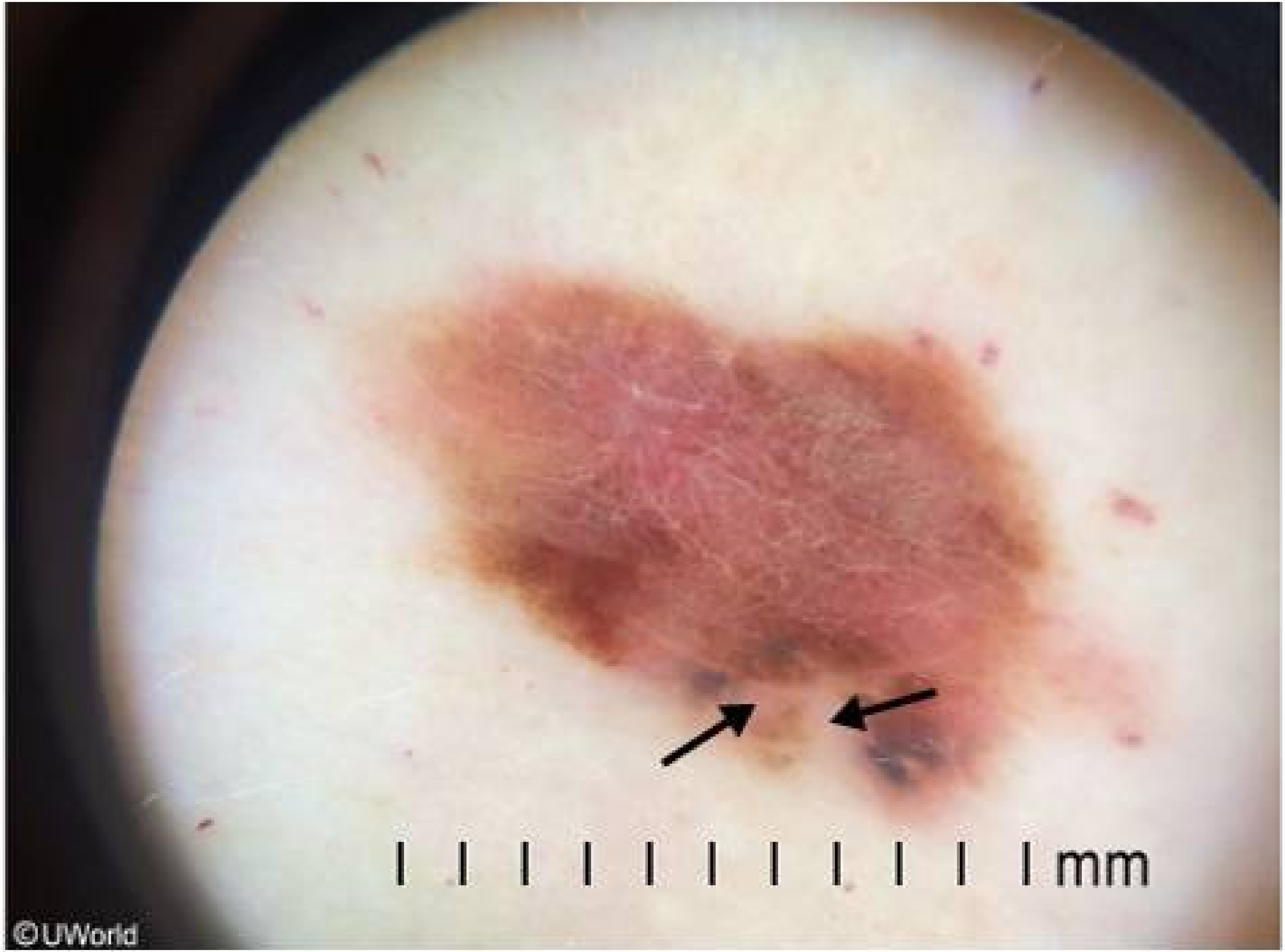




Treatment of melanoma>>

Primary treatment is excision with appropriately wide margins.
Metastatic or unresectable melanoma in patients with *BRAF* V600E mutation may benefit from vemu**raf**enib, a BRAF kinase inhibitor.

Melanoma>> melanocyte regression



Squamous cell carcinoma>>

Second most common skin cancer.

Associated with immunosuppression, chronic nonhealing wounds, and occasionally arsenic exposure.

Commonly appears on face, lower lip, ears, hands.

Locally invasive, may spread to lymph nodes, and will rarely metastasize.

Ulcerative red lesions.

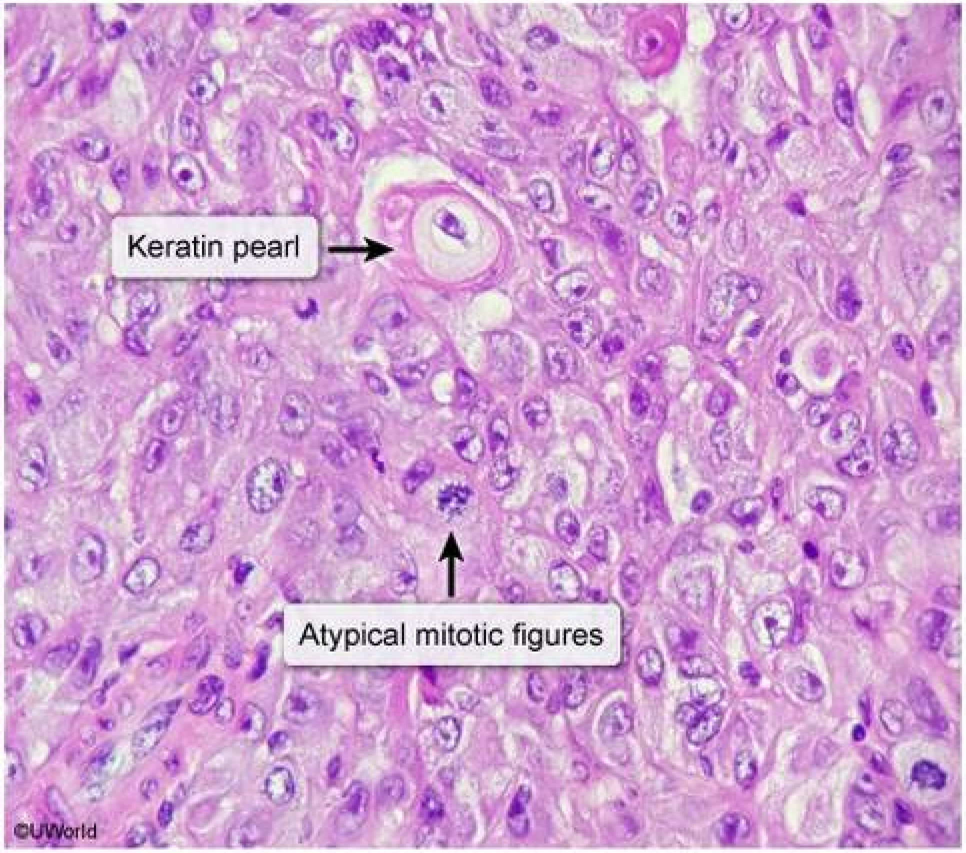
Histopathology: keratin "pearls".

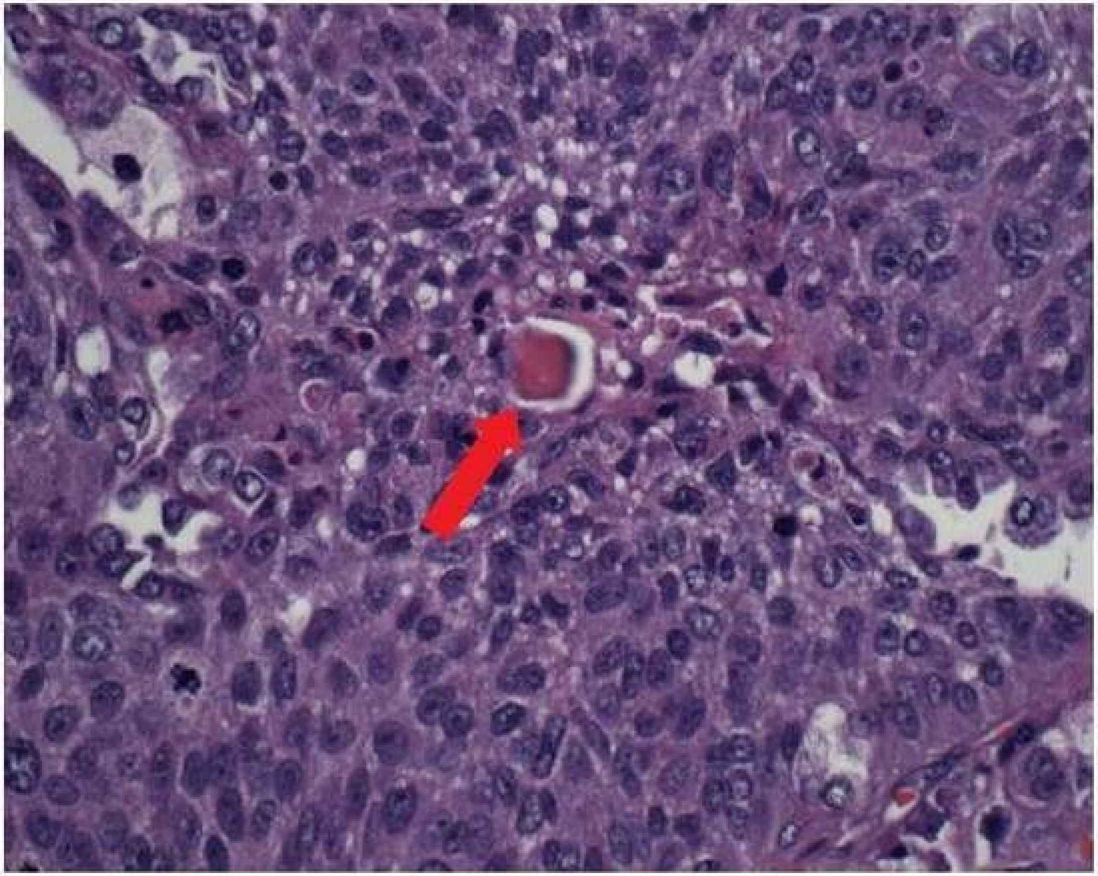
Actinic keratosis, a scaly plaque, is a precursor to squamous cell carcinoma.

Squamous cell carcinoma>>

Squamous cell carcinoma >> Marjolin ulcer

Squamous cell carcinoma>> keratin "pearls"





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